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BLAKISTON BOOKS ON AGRICULTURE

NATURE and PREVENTION of
PLANT DISEASES



One of the best and sometimes the only means of controlling destructive plant disease is the development of disease-resistant crop varieties.

The devastation of the curly top disease led to wholesale abandonment of sugar beet fields and sugar mills, an industry that was brought back to life and prosperity by the development of curly top resistant beet varieties.

In this beet field, exposed to the ravages of curly top, the center eight rows of old-type beets have been all but wiped out, while adjacent plantings of resistant varieties, at left and right, give promise of a bumper crop. (Photograph courtesy of The Bureau of Plant Industry, Soils, and Agricultural Engineering, U.S.D.A.)

NATURE and PREVENTION of PLANT DISEASES

By K. STARR CHESTER, Ph.D.

Battelle Memorial Institute
Columbus, Ohio

Second Edition



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Second Edition

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Preface

To the Student

This book is written for you. The material and manner of treatment of the subject have developed around the needs and interests of my students of the past ten years, who are really the unsigned authors. I believe that your study of the causes and prevention of plant diseases will be well worth the effort and very helpful in your future agricultural activities. I hope that in this book you will find the information that will be most useful in the prevention of plant disease and that it will serve you not only as a textbook during your college course but as a helpful reference in post-college years.

To the Instructor

This book is designed particularly for the use of students whose formal training in plant pathology is limited to a single semester or year, whose practical interest in the subject is a desire to know how to recognize, understand, and prevent plant diseases, and to whom the elementary course in plant pathology is a necessary part of the background for useful work in agriculture.

With this purpose in mind it has seemed desirable to include, as far as possible, the leading diseases of the major crops grown extensively in the United States, to emphasize the recognition of these diseases, and to indicate in some detail the latest generally approved methods of their control.

At the same time, some knowledge of the principles underlying the science of plant pathology is indispensable for an understanding of the behavior of plant pathogens and the results of their activities, and to enable the student to adapt his basic training to new plant disease situations with which he may be faced.

A book emphasizing principles, to the extent that specific information on the control of many plant diseases cannot be included, is perhaps most suited to those students for whom the first course in plant pathology is followed by advanced courses—those training for plant pathology as a profession—but such a book would fall short of the needs of students whose first course is a terminal course. At the other extreme, to limit the book to recognition and control of plant diseases might satisfy the needs

of field workers for a handbook, but would fail to give the student adequate understanding of the causes of plant diseases and the behavior of the plant pathogens.

An attempt has been made to steer a middle course between these extremes. The principles of the science are brought out in fairly detailed accounts of one or more diseases of each type. The treatment of numerous diseases of the same type is more limited, with discussion of unusual features, recognition, and control. In the closing chapters, the principles of plant pathology encountered one by one in the study of the various disease types are organized in form to serve as a unified treatment and review of the leading features of the science and as a compact source of information on disease control procedures.

These factors have made it seem desirable to limit to a minimum the treatment of such academic aspects of plant pathology as history, mycology, and parasitism *per se*. Understanding and appreciation of these aspects of the science are essential for the professional plant pathologist, but in a terminal course primarily for agricultural students the practical aspects of the science must receive first consideration.

It is held by some instructors that a textbook of this nature should minimize detail in disease-control recommendations since these are constantly changing. Provided that such a book can be revised and brought up to date periodically, it is felt that the changes in detail of control practices which occur between editions will not be so great but that they can be explained, without undue effort on the part of the instructor or confusion of the student, in the classroom and laboratory.

In the present edition preliminary treatments of the several types of causative agents of plant disease largely are transferred to the chapters dealing with each disease type, rather than considered as a whole in a preliminary chapter. The arrangement of diseases within chapters is a compromise in which the systematic relationships of pathogens are given first consideration, but with such modification as is necessary to avoid too abrupt and illogical transitions between disease types. All diseases caused by species of *Fusarium*, for example, could hardly be treated together without violation of pathologic unity. The chapter on virus diseases has been completely rearranged in groupings of the mosaic, yellows, phloem necrosis, and scaly bark types followed by the potato and stone fruit virus complexes.

Numerous important diseases that were considered very briefly or not at all in the first edition have been taken up in greater or less detail. These include apple bitter rot, sugar cane red rot, cabbage yellows, black root rot of tobacco, early blight of potato and tomato, tomato leaf mold

and leaf spot, potato ring rot and scab, aster yellows, sugar beet curly top, phloem necrosis of elm, citrus psorosis, golden nematode of potato, and witchweed. Extensive revisions will be noted in the discussions of seed treatment and spraying and dusting of fruits and vegetables. Attention is given to the latest developments in control practices, including the slurry, pelleting, and vapor-heat methods of seed treatment, the new nonmetallic organic fungicides, and innovations in methods of spraying and dusting. Some 30 new illustrations have been added. Numerous recent references have been added, all of them in English and commonly available in agricultural libraries.

The preparation of this edition has been aided by helpful suggestions from many colleagues and users of the first edition, to whom I extend thanks. Grateful acknowledgment is expressed to Dr. G. H. Collings and also to those who have aided in preparation of the new illustrations, Drs. H. I. Featherly, D. L. Fernholz, others whose names or organizations are mentioned in the legends, and to my associates, Dr. W. W. Ray and Mr. D. A. Preston, who have helped by critical reading of the manuscript.

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Contents

PREFACE.	v
1. SIGNIFICANCE OF PLANT DISEASE IN AGRICULTURE.	1
2. TYPES OF PLANT DISEASE: FUNGI	15
FUNGI.	
3. DISEASES CAUSED BY BASIDIOMYCETES: RUSTS	22
INTRODUCTION TO THE RUSTS. RUSTS OF FIELD CROPS: stem rust of grains and grasses; leaf rust of wheat; stripe rust of wheat and other gramineae; crown rust of oats; corn rust; barley leaf rust; rye leaf rust; sorghum leaf rust; flax rust; sunflower rust; alfalfa rust; clover rusts. RUSTS OF GRASSES. RUSTS OF TREES AND SHRUBS: cedar apple rust; blister rust of five-needle pines; rust of stone fruits; orange rust of cane fruits. RUSTS OF VEGETABLES: asparagus rust; bean rust; sweet potato rust. RUSTS OF ORNAMENTALS: carnation rust; snapdragon rust; hollyhock rust; other rusts of ornamentals.	
4. DISEASES CAUSED BY BASIDIOMYCETES: SMUTS	49
NATURE OF THE SMUTS. SEEDLING INFECTION SMUTS: hunt of wheat; other seedling infection smuts. BLOSSOM-INFECTION SMUTS: loose smut of wheat; brown loose smut of barley. LOCAL INFECTION SMUTS: common smut of corn; onion smut; white smuts.	
5. DISEASES CAUSED BY BASIDIOMYCETES: FLESHY FUNGI; MYCORRHIZAE.	74
Wood decay; white trunk rot; shoestring root rot; mycorrhizae; rhizoctonia disease of potatoes.	
6. DISEASES CAUSED BY ASCOMYCETES.	94
Scab of cereals; apple scab; apple black rot; apple bitter rot; apple blister canker; grape black rot; black knot of plums and cherries; elm leaf spot; sycamore anthracnose; red rot of sugar cane; iris leaf spot; sweet potato black rot; Dutch elm disease; chestnut blight; ergot of grains and grasses; powdery mildew of cereals; powdery mildew of roses; other powdery mildews; brown rot of stone fruits; cherry leaf spot; alfalfa leaf spot; anthracnose of cane fruits; black spot of roses; peach leaf curl.	
7. DISEASES CAUSED BY IMPERFECT FUNGI.	142
DISEASES CAUSED BY HYPHOMYCETES: wilt diseases: cotton wilt; tomato wilt; watermelon wilt; flax wilt; sweet potato wilt or stem rot; cabbage yellows; other Fusarium wilts; Cephalosporium wilts; Verticillium wilts; Texas root rot; black root rot of tobacco; dry-land foot or root rot of wheat; Helminthosporium diseases of barley; other Helminthosporium diseases; early blight of potato and tomato; tomato leaf mold; peach scab; Cercospora diseases; gray molds. ANTHRACNOSE DISEASES CAUSED BY MELANCONIALES: cereal anthrac-	

nose; melon anthracnose; anthracnose of beans; other anthracnose diseases. DISEASES CAUSED BY SPHAEROPSIDALES: apple blotch; *Phyllosticta* leaf spots of other hosts; rose cankers; *Septoria* diseases of wheat; *Septoria* leaf spot of tomato; celery late blight.

8. DISEASES CAUSED BY PHYCOMYCETES AND RELATED FUNGI . . . 210

Late blight (downy mildew) of potato and tomato; downy mildew of cucurbits; downy mildew of spinach; downy mildew of tobacco; downy mildew of grapes; downy mildews of grains and grasses; other downy mildews; white rusts; white rust of crucifers; white rust of spinach; sweet potato soft rot and strawberry leak; brown spot of corn; clubroot of crucifers; slime molds.

9. DAMPING-OFF AND RELATED TROUBLES. 238

Damping-off; the cotton seedling blight and boll rot complex; corn root, stalk, and ear rots; sorghum root and stalk rots.

10. DISEASES CAUSED BY BACTERIA 262

Bacteriology; pathology; fire blight of apple and pear; bacterial spot of stone fruits; bean blight and halo blight; cowpea canker; soft rot of vegetables; bacterial canker, spot, and speck of tomatoes; tobacco angular leaf spot and wildfire; bacterial blight of cotton; bacterial diseases of cereals; bacterial wilt of corn; alfalfa wilt; bacterial ring rot of potato; southern wilt; bacterial wilt of cucurbits; crown gall; common scab of potato.

11. DISEASES CAUSED BY VIRUSES 298

Significance of virus diseases; symptoms of virus diseases; properties of viruses; nomenclature of viruses; nature of viruses; tobacco (tomato) mosaic; bean mosaic; wheat mosaic; sugar cane mosaic; cucurbit mosaic; aster yellows; curly top of sugar beet; phloem necrosis of elm; citrus psorosis or scaly bark; virus diseases of potatoes; virus diseases of stone fruits.

12. DISEASES CAUSED BY PARASITIC SEED PLANTS AND ALGAE; EPIPHYTES. 340

Parasitic seed plants; dodder; mistletoe; witchweed; parasitic algae; epiphytes.

13. DISEASES CAUSED BY NEMATODES OR EELWORMS. 353

Root knot; the sugar beet nematode; wheat and rye nematode; stem and bulb nematode; golden nematode of potato; root lesion nematode; nematodes: some general considerations.

14. PHYSIOGENIC DISEASES 368

DISEASES DUE TO UNFAVORABLE SOIL CONDITIONS: Lack of necessary chemical substances; excess of soil chemicals; water deficiency; excess of water and irregularity of water supply. DISEASES DUE TO UNFAVORABLE AIR RELATIONSHIPS: Lack of oxygen; poisonous gases. DISEASES DUE TO UNFAVORABLE TEMPERATURES: Heat injury; cold injury. DISEASES DUE TO UNFAVORABLE LIGHT CONDITIONS: Insufficient light; excessive light.

15. THE METHODS OF STUDYING PLANT DISEASES 393

Field observations; plant disease surveys; collections; preservation of specimens; laboratory examination of specimens; identification of organisms; investigating the causes of diseases; studies on epiphytology; studies on disease control; literature review.

16. ENVIRONMENT AND PARASITIC DISEASE.	412
Effect of temperature on parasitic disease; effect of moisture on parasitic disease; effect of wind on parasitic disease; effect of light on parasitic disease; effect of soil on parasitic disease.	
17. ETIOLOGY AND EPIPHYTOLOGY OF DISEASE	419
ETIOLOGY: Names, history, and life-history of pathogenic organisms. EPIPHYTOLOGY: Epiphytotics due primarily to weather conditions; to unnatural culture; to the introduction of disease; to plant breeding; the grand cycle of disease; origin of plant diseases; prediction of epiphytotics.	
18. PRINCIPLES AND PROCEDURES IN THE CONTROL OF PLANT DISEASES; CONTROL OF PLANT DISEASES BY REGULATION	433
Control procedures; control by regulation; biological aspects of quarantines; economic aspects of quarantines; mechanism of plant quarantines; present-day quarantines; efficacy of quarantines.	
19. CONTROL OF PLANT DISEASE BY INDUCING RESISTANCE	442
ECONOMIC SIGNIFICANCE OF THE DEVELOPMENT OF DISEASE-RESISTANT CROP VARIETIES. SOME BASIC CONCEPTS OF PLANT IMMUNOLOGY. NATURE OF DISEASE RESISTANCE AND IMMUNITY: mechanical defenses; chemical defenses; functional defenses; hypersensitivity; acquired immunity; complexity of the factors that determine resistance. CONTROL OF PLANT DISEASES THROUGH THE USE OF RESISTANT VARIETIES: the need for this type of disease control; requirements for an acceptable resistant variety; accomplishments in the development of disease-resistant varieties; genetics of disease resistance in plants; limitations of breeding for resistance.	
20. CONTROL OF PLANT DISEASES BY CULTURAL METHODS.	457
NONINFESTED SOIL: uninfested soil; disinfested soil. NONINFESTED SEED: uninfested seed; disinfested seed. CARE OF THE CROP: choice of site; preparing the soil; soil amendment; seeding; water supply; handling; harvesting; storing. REMOVAL OF UNDESIRABLE PLANTS OR PLANT PARTS: weed control by clean culture; eradication of wild hosts; eradication of alternate hosts of rust diseases; roguing; removal of infected plant parts; removal of the body or reproductive parts of the pathogen. PROTECTION OF THE GROWING CROP WITH FUNGICIDES: spraying versus dusting: combination methods; sprays and spraying; dusts and dusting; hazards of dusting and spraying; unusual chemical control practices. CURE OF DISEASED PLANTS: heat treatments of plants for nematode control; heat treatments for viruses; therapeutic treatments of trees.	
GLOSSARY.	493
INDEX.	503

Chapter 1

Significance of Plant Disease in Agriculture

Waist deep in a sea of ripening wheat stand two men, and they mark a turning point in American agriculture. The man in overalls dejectedly pulls a few stalks from the soil. The stems are cracked and dried, stained with red and black streaks. He breaks off a head of grain and rubs it between his palms, and as he blows the chaff gently away there remain in his palm a few pitifully shriveled kernels. Many of the stalks have broken over and fallen below the reach of binder or combine. The field that just a few days ago gave promise of 40 bushels to the acre, today will hardly yield the expense of harvesting.

This is the grim side of black stem rust, the scourge of wheat farmers in every land. The scene, which has taken place countless times in American wheat fields, is a classic one which had its prototypes 4,000 years ago in the grain fields of the ancient Hebrews.

What will this mean to the man in overalls? Perhaps another postponement of the children's chance for education; perhaps failure to meet the payments on the nearly paid-up farm; perhaps this year will mark the beginning of the long, sad back-trek from combine to binder, from tractor to mules, from a square mile of rich, flat bottom land to a quarter section of eroded hillside—back from wheat, which takes machinery, to cotton or corn, which you can raise if you have a mule and a family, and so on back to working for the insurance company or subsistence relief.

That is the dark side. But what about the other man beside the man in overalls? He is the county agricultural agent. He is saying something to this effect: "You don't need to put up with this loss another year. The men at the experiment stations have been working to breed varieties of wheat that are resistant to the stem rust fungus. They have been able to combine rust resistance with the other qualities we need in wheat—high yields, drought and cold resistance, and good milling and baking qualities. Jim Beard, out west of town, has been growing one of these varieties, and it is making 37 bushels to the acre this year. You can get some of that wheat for seeding, and be ready for rust another year."

Because disasters like this occur today and often mean the difference between success and failure in agriculture, and because many such disasters can be averted by timely application of simple preventive measures, some acquaintance with the science of plant pathology is indispensable to agricultural workers. The purposes of an introductory course in plant pathology, and the purposes of this book, are to enable you to become acquainted with the principal types of plant diseases, to learn of the principles of plant disease prevention and their practice in controlling important plant diseases that are injuring farming activity today, to have some background of understanding of the essentials of plant disease and its control, so that when you are faced with an unfamiliar problem in plant pathology you will be aided in solving it by your familiarity with related problems, and finally, to become acquainted with the as yet unsolved problems of plant pathology and with the efforts the United States Department of Agriculture and the state experiment stations are making to devise means for combating plant diseases.

You are studying a comparatively new science, that of plant disease. It is only a few decades since plant pathology came into being. Some of the pioneer plant pathologists, founders of the science in America, are still vigorously carrying on their warfare against plant disease, setting a stimulating example to their army of younger followers. But neither plant diseases nor their prevention by empirical or intuitive recipes constitute problems only of today. Long before the appearance of civilized man the agents of disease were leaving petrified thumbprints in the fossils that tell us of the leaf spot diseases and other ailments of prehistoric vegetation. Among the earliest written records of man, the unmistakable complaints of blights, mildews, and plagues show us clearly that plant disease has shadowed the agricultural path of man since he first scratched the soil with a pointed stick and planted seed. The Old Testament tells us of plant diseases visited upon man in punishment of his transgressions. Three hundred years before Christ, Theophrastus, the Father of Botany, was familiar with the plant diseases of his time, and in his writings we can recognize many of our plant troubles of today such as scorch, rot, scab, and rust. So formidable were the cereal rusts in those early days that the Romans evolved a rust God, Robigus, whom they annually honored as a means of rust prevention.

As ancient times gave way to the intellectual darkness of the Middle Ages, these early sparks of understanding of plant disease were all but extinguished by the superstition and avoidance of reason that overshadowed that period. Plant diseases continued to take their toll from the European peasant and landowner, but we learn little of them save that

from time to time great epiphytotics¹ occurred, attended by disaster, famine, and migrations, and historical documents of the early days tell us of entreaties to the deity to ward off the evil blights, of tragic suffering and death from the "holy fire" which we now attribute to the eating of ergot-diseased grain, of the suffering and famine in Ireland when disease destroyed the potato crops in 1845-1847 and drove many of the Irish people to America, and of the powdery mildew which wiped out the wine industry of Madeira and forced the population of that little island back to their ancient occupations of growing sugar cane and gathering cochineal.

The story of the Irish potato blight is the story of a microscopic fungus which wrought havoc in Europe equalled by few of Europe's many wars.

The potato was first brought to Europe around 1588 from the Andean hinterland of South America where it had long been revered as emblematic of fertility, and had even been the inspiration for mutilation and human sacrifice. Thanks to the efforts of Sir Walter Raleigh and many other enthusiasts, the potato soon won its rightful place as a leading source of carbohydrate food not only throughout all of Europe from the Mediterranean to northernmost Scandinavia, but in northeastern North America as well. In its migration from South America the potato had left behind its most serious agents of disease and for 200 years or more it enjoyed comparative freedom from disease. But in the early half of the nineteenth century disturbing reports of potato failures began to appear. In ever-increasing intensity, a plague of the potato fields was laying waste the crops of whole communities.

In 1845 the crisis was reached. With unbelievable destructiveness the potato blight devastated millions of acres in Europe, the United States, and Canada. So sudden and so complete was the catastrophe that in only a few days fields with every promise of abundant harvest were transformed into blackened wastes of vegetation overlying foul and putrifying masses of rotten tubers. This was not a local problem; nor was it limited to a few fields. Wherever potatoes were grown the tragedy was repeated, bringing in its wake privation, then starvation or the fever that inevitably follows malnutrition. In Ireland alone, a quarter of a million people fell victim to the famine, and many others migrated to America, becoming the nucleus of the Irish-American population of the United States. The tragedy of the potato blight is a dramatic story that is well worth reading in detail in Rolfe and Rolfe's "Romance of the Fungus World," the novel "Famine" by Liam O'Flaherty, or Large's "Advance of the Fungi."

Like most tragic experiences of mankind, the potato blight was not

¹ The name given to a destructive outbreak of plant disease; comparable to epidemics of human disease or epizootics of animal disease.

without some benefit. In the nineteenth century science was rapidly throwing off the stupor of the Middle Ages; the chains of superstition that so long had bound and suppressed creative thought were rusting away. The intellectual geniuses, Louis Pasteur and Robert Koch, were performing the first crucial experiments that were to open up the vast field of modern research on contagious disease. Charles Darwin was revolutionizing biology and philosophy with his keen deductions on organic evolution. Von Liebig was laying the foundations of modern agricultural chemistry. The stage was set for the first fundamental discoveries on the nature and control of plant disease, and the catastrophe of the potato blight forced the attention of master minds to the solution of this and related problems in plant pathology. Out of the labor pains of Europe, racked by the potato blight, was born modern plant pathology, the science of plant disease. The brilliant young German, Heinrich Anton de Bary, stared at the dying potato leaves through his primitive microscope, saw the green leaf cells in the clutches of the sinuous, pallid fibers of the fungus, and its myriads of wind-driven spores, proved that the fungus was the sole cause of the blight, and paved the way for Millardet a few years later to give humanity an effective weapon against any future recurrence of the blight—Bordeaux mixture.

The story of Bordeaux mixture itself is worth the telling. According to tradition, a farmer in Médoc, France, had a vineyard that bordered the highway. Passers-by are alike the world over and, to the despair of the farmer, the wayfarers could not resist the luscious bunches of ripening grapes just over the fence. In a moment of inspiration, the farmer decided to take steps. He went to the barn, and his eye falling on a sack of lime, he made a milky broth to splash on the vines. As the mixture didn't look repulsive enough, he threw in a shovelful of bluestone. This accomplished, he spattered it over the vines, posted a "Poison" sign and awaited results. History does not tell us whether the wayfarers were deterred by the farmer's ingenuity, but it does recall that Millardet came past the vineyard, noted that only the sprayed grapes had escaped the destructive mildew disease, learned of the spray so accidentally applied, tested its efficiency against fungus diseases of the vine, and gave us the effective protection against future outbreaks of both vine and potato blights which we now know as Bordeaux mixture.

Man has a tendency to learn things the hard way. It took another epiphytotic which has practically exterminated one of our finest forest trees, the American chestnut, to establish the science of plant pathology in America. The chestnut blight fungus was a foreigner that sneaked into America from Asia. Starting its deadly work about 1904, it spread swiftly,

destroying every tree in its path. Today there hardly remains a chestnut tree in the great eastern forests which were once dominated by this tree. This disaster taught us what might be expected from unwelcome foreign pests; it was largely responsible for the establishment of the National Plant Quarantine Act of 1912.

Today, new and potent enemies of our cultivated plants are coming to the attention of growers and scientists. The Dutch elm disease for a while threatened to exterminate the American elm, as it destroyed the elm in many parts of Europe. The stately elms in the long avenues in the royal gardens at Versailles were mature trees in the heyday of the pre-Revolution French court. Only a few years after the Dutch elm disease appeared, the avenues were lined with dead and dying trees, nearly all sacrificed to the elm disease fungus (Fig. 1). Thanks to our lesson from the chestnut blight and to energetic eradication of diseased elms in America, the elm disease has been brought under control. This has been complicated by discovery in 1931 of a destructive wilt disease that kills elm trees, furnishing habitats of dead wood in which breed the beetles that spread the Dutch elm disease. Nor was this the end of the threat to the American elm. In 1938 came the first report of still another and even more menacing elm disease, a virus disease which rapidly spread in the Mississippi valley. In 1944 alone, 20,000 elms were killed in Dayton, Ohio, and 10,000 in Columbus, and the next year even greater numbers were killed as the disease rapidly continued its deadly march west and north. The sycamore and the oak are threatened by other virulent disease organisms. During the last few years the native persimmon in the south central states has fallen victim to a fungus which equals the chestnut blight organism in its virulence. This may be welcome news to farm lads who have worked in the hot sun grubbing weedy persimmons out of the pasture. The editor of a leading Southern agricultural journal wrote: "I am delighted to hear the persimmon disease has arrived in the Southwest." But there is also much to be said in favor of the persimmon—its delicious fruit, its value in feeding 'possums and other desirable wild life, its usefulness in preventing soil erosion, and to a limited extent the use of its exceedingly hard wood in such articles as the heads of golf clubs. Already large areas have been depleted of persimmon by the ravages of the disease and a real possibility exists that this tree may emulate the fate of the dodo, the heath hen, and the American chestnut.

Few of the main groups of crop plants are free from occasional but disastrous attacks of disease. Among the fruits may be mentioned fire blight which caused "one of the greatest industries of the San Joaquin valley to vanish like a dream" when 500,000 pear trees were killed by the



FIG. 1. The threat of the Dutch elm disease. (*Top*) A scene along an elm-shaded village street. (*Bottom*) As the same street would appear if the elms were destroyed. This has already happened in many parts of Europe, but prompt quarantines and energetic eradication of diseased trees have prevented the disease from becoming general in America. (Courtesy, M. A. McKenzie, Mass. Agr. Exp. Sta.)

disease within a few years. In the rich fruit section of New York State a new virus disease of peaches has broken out in epiphytotic form, promising to be even more destructive than any of the other numerous virus diseases of this tree. In the tropics banana plantations cannot be permanent. Invariably they become infested with the "Panama disease" after a few years. There has been no escape; the infested plantations have been abandoned, and the industry has continually pushed on to new plantings in virgin soil.

Among vegetables, the ravages of the potato blight are seconded by those of watermelon wilt, at first welcomed as nature's way to maintain price levels by restricting production, but soon wiping out the melon industry in important sections of Florida, Iowa, and California (Fig. 2). The potato itself has been plagued recently by outbreaks of new or unusual diseases. Yellow dwarf, which is much more destructive to the potato plant than its many other virus diseases, has lately assumed national importance, causing total loss in numerous fields over a broad area. The golden nematode, a pest of major importance in Europe, has arrived and established itself on Long Island, cutting yields of susceptible potato varieties by 70 per cent and arousing serious concern lest it spread to the main potato growing areas. Purpletop wilt, a formerly obscure but dangerous potato disease, has been found to be caused by the aster yellows virus, now become well known to vegetable growers for its destructive-



FIG. 2. Typical of many watermelon fields laid waste by the wilt disease, this commercial field of Dixie Queen melons shows great bare areas where plants have succumbed. Until the development of wilt-resistant varieties of watermelons, such fields were henceforth useless for the cultivation of watermelons, since the wilt fungus remains virulent in the soil for many years. (Courtesy, M. N. Walker, Fla. Agr. Exp. Sta.)



FIG. 3. Texas root rot destroying a stand of alfalfa at Sacaton, Arizona, in 1941. The white line of dying plants marks the steady advance of the disease, leaving behind it a few surviving alfalfa plants and a tangle of weeds. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

ness to carrots; onions, spinach, lettuce, cabbage, celery, and parsnips. Southern spinach fields, source of much of the national crop, are being ravaged increasingly by formerly unimportant diseases—blue mold and white rust.

The story is the same with field crops. Flax always has been a pioneer crop, moving on to virgin areas and leaving behind a trail of "flax-sick" soil, infested with the flax wilt fungus, soil upon which susceptible flax cannot again be grown for many years. Texas root rot has rendered great areas of the Southwest unsuitable for culture of cotton, alfalfa, and many other crops (Fig. 3). The disease causes a loss in Texas of 300,000 bales of cotton a year, and in addition, attacks more than 2,000 other species of plants, aggregating a total loss from this disease, in the seven states affected, which reached \$150,000,000 in 1937. In the Sudan and Nigeria is another serious threat to cotton—leaf curl—a virus disease which can steal as much as 300 pounds of cotton per acre. It cannot be predicted whether this virus may be brought to America, nor can the potentialities of its introduction be gaged, but in Africa it is regarded as highly menacing. Finally, no account of epiphytotics in field crops can omit mention of the cereal rusts. Rust is always with us, and now and then, when the weather is suitable, it rages northward from the Great Plains to Canada, leaving in its wake millions of acres of wasted grain. The epiphytotic of 1935 destroyed a quarter of the national wheat crop, a total of 160,000,000

bushels, and in North Dakota and Minnesota 60 per cent of the wheat crop was sacrificed to stem rust.

This is the spectacular side of plant disease, the great epiphytotics that are so often followed by privation, suffering, loss of homes and farms, even famine, migration, or abandonment of farming. Tragic as these outbreaks are, they are surpassed by the multitude of less dramatic but more prevalent ailments of plants. One would not be inclined to think of the maple tree as commonly suffering from disease. Yet, not less than 54 contagious diseases affect this tree. There are known to be at least 77 diseases of wheat in America, 112 of corn, and nearly 200 of the apple. In the potato there have been described more than 50 virus diseases, exclusive of other types, and while this probably represents some duplication, there are doubtless from 12 to 20 such diseases of the potato in addition to many fungus and bacterial diseases.

The losses from most of these lesser troubles do not reach high percentages. Like the common cold, they are always with us. They kill a plant here or there or reduce production by 1 per cent or 5 per cent, and the grower, if he is even conscious of them, has come to look upon them as necessary evils to be accepted with philosophy as part of the gamble in farming. The average farmer usually is not concerned with any plant disease until the loss reaches 15 per cent of the crop, and he rarely takes steps to prevent it until the loss mounts to 25 per cent.

American business has learned the importance of little leaks. The only difference between the borrower and the banker is a little difference of 3 or 4 per cent. Money safely invested at 4 per cent interest is a stride toward prosperity, and a crop loss of the same amount equally represents retrogression.

How many farmers realize that a small percentage of loss in the field represents a much larger loss, perhaps all, of the profit? To be specific, take the case of a farmer with a quarter-section in wheat, and assume that under disease-free conditions his average yield is a conservative 25 bushels to the acre, or a total of 4,000 bushels. The harvest return is divided into two elements: a part, usually most of it, must be paid out to cover all the costs of production of that crop; the remainder is the farmer's profit, and may be applied to maintaining and improving his standard of living and of farming. Under normal circumstances the 4,000 bushels would be used somewhat after this fashion: use of the land, 40 per cent; seed, 3 per cent; labor, 12 per cent; machinery and maintenance, 20 per cent; insurance, 5 per cent; leaving a profit of 20 per cent based on disease-free conditions. The loss from diseases in the American wheat crop for the period 1919 to 1937 averaged slightly more than 10 per cent per year. Let us assume

that our potential 4,000 bushel wheat crop was subjected to disease to this extent, and that 10 per cent or 400 bushels were lost through disease. All of the costs of production are unchanged; it still cost 3,200 bushels to produce the 3,600 bushel yield. The bills could not be paid with the diseased grain or that which failed to materialize. Ten per cent disease in the field did not strike the farmer as an unusual or serious loss; yet, the 10 per cent field loss cost him one-half of his profit.

In times of prosperity, of favorable crop weather, and high prices, such losses, needless though they may be, can be borne by the farmer without hardship and need not be a serious concern of the layman, but in the recurrent periods of low agricultural prices the farmer's plight becomes acute. Although disease losses are felt at all levels of farm prosperity they are most acute as they affect the marginal farmer; to him the percentage of loss is the back-breaking straw, the difference between survival and failure.

We hear much of the misfortunes of the American farmer as compared with the greater security and prosperity of the American business man. We blame this difference on many factors, but is not a part of the explanation in the differences in methods between the two? To the business man a loss of 1 per cent in his industry through waste is a vital loss, one to be corrected. The story is told that Mr. Rockefeller, in an inspection of one of his factories, noticed a machine dripping solder on oil cans. He asked and found that the superintendent had never tested the exact amount of solder needed. Mr. Rockefeller counted and found that the machine was applying 39 drops of solder per can. An experiment was devised on the spot; it was discovered that 38 drops would suffice. In a year's time the concern had been saved \$10,000 worth of solder and time through this slight economy. No business a fraction as wasteful as the average farm could survive without subsidy in the face of its competition. When the American farmer learns to regard his farming as the business man regards his business, we venture to predict that the need for farm relief and crop subsidy will be materially decreased.

We are witnessing many drastic changes in the pattern of American agriculture, and each of these brings the possibility of new crop disease hazards. Irrigation farming, which is so rapidly increasing, requires a shift from dry land crops to more valuable specialty crops, particularly fruits and vegetables. High returns must be received to pay the cost of irrigation, and if these new crops become decimated by disease, as can easily happen under moist conditions and culture by growers who are inexperienced with the newer crops, tragic failure of the new enterprises can result. Many other attempts are being made to grow crops where they have never grown before: to eliminate the poverty farming characteristic of

many agricultural areas, to rebuild depleted soil, to supply truck crop and ornamental plant needs about large cities, to replace single crop farming by diversified farming, to cultivate exotic crops, and to supply distant markets through modern rapid transport. Mechanized farming is radically changing the manner of growing crops and the varieties grown. Each of these changes disrupts the balance between plant parasites and their host plants, and in many cases they are followed by new, destructive outbreaks of plant disease for which the farmer must be prepared if his new agricultural venture is to succeed.

When watermelon wilt first appeared in Florida melon plantings a few growers reported the new disease that was killing the vines, and the Florida Experiment Station undertook to find means of checking the disease. The attitude of some of the growers in the early 1920's savors strongly of more recent agricultural philosophy. They said: "If this disease is eradicated, there will be a surplus of watermelons; the price will be lowered, and our profits will lessen. We do not approve of efforts to prevent wilt." But wilt is not a disease that can be trifled with. A few years after its introduction, affected land became useless for melons; losses of 90 per cent of the crop were not uncommon. The industry found it necessary to move on to new land expensive to clear. The abandoned land went back into scrub-oak since it was not suitable for other crops.

Now a new thought crystallized in the growers' minds. A profitable industry was seriously threatened. They carried their problem to the Florida Legislature, and funds were appropriated for a study of wilt. At the Florida Experiment Station a watermelon wilt project was initiated, and within a few years Dr. Walker of that Station announced that the "Leesburg," a new and desirable wilt-resistant melon was available to growers. Further improvements have followed, in particular the development of better shipping qualities in the resistant melons; gradually the abandoned land is being reclaimed as it again becomes attractive to growers, and with it returns the prosperity that attaches to successful production even in a competitive field. The same story has been repeated in sandy southeastern Iowa, in California and Texas, and in the Rush Springs area of central Oklahoma.

At this point we meet the challenge of modern agricultural philosophy. Is our farm prosperity dependent upon reducing production? And is toleration of disease losses an intelligent way to reduce over-production? As to the first question, opinions justifiably may differ. To those, who like Joseph, look forward to the seven lean years, any interference with production may ultimately work hardship. The others, the sponsors of reduced production, insist at the same time on uniform production, the

"ever-normal granary." So long as plant disease is out of hand we have no control of production; the ever-normal granary is the shuttlecock of fungus and weather.

Decreased production, if it is to be equitable and avoid hardship, must divide the load of production and the profits of production as equally as possible among growers. If plant disease subtly and evenly lowered production on all farms it might be tolerated in the interests of reduced production. But it does not. Each year it leaves some farms unscathed, "overproductive," while others are ruined. A short-sighted Texas wheat grower might rejoice at wheat failure in the Dakotas, feeling that he gains, through the destruction of competing crops. Actually, the loss of a considerable part of the United States crop has less influence on the wheat price than many other factors quite unrelated to production. Next year the reverse may be true, and it will be the Texas farmer who suffers. In farming as in every industry, annual extremes of profit and loss are much more difficult to bear than a uniform medium level of return. Such extremes may be due to uncontrollable factors, such as rainfall and temperature, and this is all the more reason for minimizing the production extremes due to controllable factors, such as many insects and diseases, to buffer and partly offset the suffering and waste caused by these extremes.

But consider the years of "normal production." During those years is not a moderate percentage of plant disease useful in preventing overproduction? Let any academician who vouchsafes to ask that question come South, close to the land in the fall. Schools have been closed to let the children help with the cotton picking. The women, too, and the old people have wrapped their knees with burlap and crawl on hands and knees, a 50-lb., 10-ft. sack tugging at one shoulder.

Cotton diseases are causing a loss of one-fifth of the crop annually. Prevention of these diseases, many of which can be controlled, does not need to mean a 20 per cent increase in American cotton production. Might it not better mean a 20 per cent reduction in the labor of planting, chopping, and picking, some release of children and women from this grinding drudgery, a release of 20 per cent of depleted cotton land for a program of soil restoration? Whichever philosophy we accept, the moral is the same: the prevention of waste from plant disease does not mean suffering from overproduction; on the contrary it means an opportunity for improving the lot of the farmer by aiding to buffer him against the shock of sudden and unpredictable crop losses, and by giving him some measure of alleviation of the economic and social burden under which he labors.

This, then, is the challenge of American agriculture to the American

scientist: "You can see our problem; we are calling on you to help us," a challenge blended of thousands of pleas to the Federal and State Experiment Stations.

And how are the scientists meeting this challenge? One of the newest branches of science, plant pathology, already has enlisted a thousand or more specialists. In Washington, at the state colleges, in private institutions and plant industries these men are devoting their lives to a crusade against plant disease. Much has been accomplished; against many destructive diseases highly effective chemicals of prevention have been found: sprays and dusts for fruit and vegetable crops, simple and inexpensive chemical dust treatments for ridding seeds of the germs of disease, chemicals for disinfecting soil, benzol vapor for protecting tobacco seedlings from mildew, fermentation acids for sterilizing tomato seeds, and a host of others. Better, because they are simpler, are the measures of disease control which depend only upon slight changes in the ways of cultivating plants: changing the date of planting to favor the plant and inhibit its parasites, rotation of crops to starve the parasites out of the soil, farm sanitation to destroy the breeding and hiding places of plant pests, to mention only a few of these. Best of all are the scores of new varieties of plants, joint contribution of the plant breeder and the plant pathologist, varieties that are innately resistant to the attack of parasites and at the same time desirable commercial types. There are, for example, the wilt-resistant Bison flax, Pan America tomato, Dixie and Rowden cottons, Kleckley watermelon, and Ranger alfalfa. New rust resistant small grains are coming to the fore and enormous acreages have already been planted to some of these. Resistant varieties have brought back to profitable existence agricultural enterprises that were on the verge of collapse because of such diseases as sugar cane mosaic, sugar beet curly top, and Granville wilt of tobacco. Resistant varieties have made it possible to grow crops successfully in areas where, because of disease, this could not be done before, as in cereal culture in the Coastal Plain of Texas. The newer varieties often combine in a single productive and well-adapted crop resistance to several diseases at once, for example in the tobacco Ky. 34 which is resistant to black root rot, wilt, and mosaic disease; Clinton oats that are resistant to two rusts, smuts, the new and deadly Victoria blight, halo blight, and lodging; the Arkansas Fortuna rice with resistance to three major diseases; new potato varieties resistant to late blight and certain virus diseases, and many others. For some soil-borne diseases, resistant understocks have been found, on which susceptible tops may be safely grafted.

The story of this winning fight against plant disease is a gripping story of onward marching in the face of many obstacles. There have been

failures, and much remains to be accomplished. Many plant diseases still resist efforts at their control. With others, we have methods for prevention but they are costly, difficult, or disagreeable. But as the American farmer moves on into the task implied by the economic stress of today, he will have in the background the hundreds of scientists, quietly working with him, providing him with the knowledge he needs to lighten his own economic load and permit him to produce the raw products that America needs, amply, efficiently, and economically.

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Chapter 2

Types of Plant Disease: Fungi

In its broadest sense, disease in a plant is any alteration that interferes with its normal structure, functions, or economic value. Thus defined, plant diseases may be classified as follows:

I. Physiogenic Plant Diseases. These abiotic or nonparasitic diseases are caused by such environmental disturbances as deficiencies of food materials in the soil, excesses of soluble salts in the soil, unfavorable water, air, and light relations, unfavorable temperatures, injurious chemicals, and mechanical injuries. These subjects are treated at length in courses and books on agronomy, horticulture, and plant physiology, and are here considered only very briefly.

II. Diseases Caused by Viruses. These infectious principles are invisible with the microscope, but are highly contagious, causing many diseases of animals, such as sleeping sickness, infantile paralysis, smallpox, and rabies, and important diseases of plants, such as the mosaic diseases of tomato, potato, and cucumber, the yellows diseases of peach and aster, curly top of sugar beets, and many others.

III. Diseases Caused by Parasitic or Predatory Animals.

1. **PROTOZOA.** Certain flagellates are found in the latex of milky plants, but important cases of parasitism by protozoa in plants are rare or lacking.
2. **NEMATODES OR EELWORMS.** A few species are responsible for important plant diseases, and are included in the field of plant pathology.
3. **MOLLUSCS** (snails, slugs, etc.).
4. **ARTHROPODS**, including insects, mites, and millipedes. These are treated in entomology.
5. **VERTEBRATES**, particularly rodents and birds. Usually these are considered in connection with entomology or wild-life.

IV. Diseases Caused by Pathogenic Plants.

1. **PARASITIC FLOWERING PLANTS.** Dodder, mistletoe, and a few flowering plants parasitic on the roots of other plants.

2. **ALGAE AND LICHENS.** With a few pronounced exceptions these rarely cause disease.
3. **BACTERIA.** Causes of many important plant diseases, such as corn wilt, alfalfa wilt, cotton blight, fireblight of fruits, soft rot of vegetables.
4. **FUNGI.** Familiar examples of *fungi* (singular, *fungus*) are blue, green, and black molds common on decaying foods, mushrooms, rusts, smuts, and mildews. Of the thousands of species of fungi, most are harmless saprophytes, feeding on dead animal or plant materials, and benefiting man by breaking down these organic substances into their simpler elements and restoring them to the soil. Some fungi are parasitic on insects and nematodes, aiding in the control of these pests; others are destructive to fish; a few cause disagreeable skin diseases in man, as "athlete's foot," and "ringworm"; and many are parasitic on plants, producing the great majority of parasitic plant diseases.

Fungi

Structure of Fungi. The body or thallus of a fungus consists of delicate threads or *hyphae* (singular, *hypha*) which are often so numerous as to form a branched system of threads called *mycelium*, familiar in the cottony masses of hyphae of the common bread mold. These are white, or less commonly red, brown, yellow, or greenish, but they contain no chlorophyll and are unable to manufacture their own food. This must be obtained from dead organic matter or stolen from living plants or animals.

The mycelium of different species of fungi shows great variation in appearance and structure. The hyphae may be divided into cells (*septate*) or *nonseptate*, coarse or fine, aerial or submerged, stiff or flexuous, and may exhibit different types of branching. These characters often are useful in identifying plant disease fungi.

At times the hyphae join in parallel to form long, ropelike strands called *rhizomorphs*, by which the fungus makes its way for considerable distances through the soil or along or under the bark of woody plants (Figs. 33, 75). At other times the hyphae may form hard, compact, rounded or scaly masses analogous to a tightly compressed ball of yarn, called *sclerotia* (singular, *sclerotium*) which serve as resting bodies to carry the fungus through hot, cold, or dry periods (Figs. 35, 77). By means of sclerotia some fungi can survive for many years in soil, plant refuse, or seed.

While fungi often can reproduce by means of broken-off bits of mycelium or other vegetative means, their common method of reproduction is by *spores*. These are one- or several-celled, microscopic bodies,

analogous in function to seeds of higher plants in spreading and perpetuating the species. In adaptation to their remote chance of reaching and successfully infecting susceptible host plants, the spores usually are produced in prodigious numbers. Spores vary greatly in size, shape, color, thickness, and structure of the spore wall, appendages, appearance of contents, and manner of production and germination, and the identification and classification of fungi are based largely on the character of spores, the *fruiting bodies* that contain them, and the spore-bearing hyphae on which the spores are produced.

Spores may be borne on hyphae more or less at random over the surface of mycelium, on clusters of hyphae that are grouped together like wheat stalks in the sheaf, or inside fruiting bodies in the form of cups, flasks, mushrooms, brackets, etc., which, in turn, exhibit many distinct characters that serve to distinguish the genera and species of fungi (Figs. 28, 36).

Many fungi have sexual reproduction. The mating of distinct fungi, sometimes referred to as "plus" and "minus" rather than male and female strains, or of opposite-sexed organs on a single fungus, may be prerequisite to spore production. The manner of production of sexually formed spores is the basis of classification of fungi into their main groups, *Phycomycetes*, *Ascomycetes*, and *Basidiomycetes*, and a fourth group in which sexually-produced spores have not been found, is termed the *Fungi Imperfecti* or imperfect fungi.

Fungi which produce spores sexually also often have one or more other types of spores, formed without sexual processes, or imperfect spores. In many cases these are called *conidia* and, like the sexual spores, they may be borne at random over the surface of mycelium or be in more or less elaborate fruiting bodies. Usually they are adapted to being borne to distant points by the wind, but some can swim about actively, and others have no special means of dissemination but are washed away by rain or drop to the ground where they may stay or be moved about by man, animals, farm machinery, and water. A special type of asexual spore, the *chlamydospore*, is simply an enlarged hyphal cell, occurring singly or in groups in hyphae, with a thick wall favoring the survival of the chlamydospores in soil or plant debris (Fig. 68).

Nutrition of Fungi. The majority of fungi are saprophytes, living in soil or on dead organic matter. They can secrete powerful enzymes which convert the complex organic substances into simpler foodstuffs. In this they perform an indispensable service to the living world, restoring the elemental food materials to use by plants. Were it not for their activity the world would soon become an unlivable charnel house of undecayed plant and animal remains.

Fungi have no special means of feeding. The food materials with which they come in contact are absorbed directly through the hyphal walls. Some of the saprophytic fungi produce powerful poisons which aid them in their saprophytic struggle for existence by destroying other organisms with which they come in contact—the fungus *Penicillium* with its germ-killing poison penicillin is a well-known example.

Even more highly adapted to their mode of life are the disease-causing plant parasitic fungi or *pathogens*. These must obtain their food by invading living plants, called the *host plants*, overcoming their various defense mechanisms, and wresting from them a livelihood which often eventually kills the host plants.

Infection by Fungi. If infection is through the agency of fungus spores, the spore must be brought in contact with the plant by wind, rain, insects, etc. Under favorable conditions of moisture and temperature the spore germinates, usually within a few hours, extending outward a hypha (*germ tube* or *infection thread*) which may either grow into the plant through a natural opening (stoma, lenticel) or a wound (insect sting, mechanical injury), or it may force its way in through unbroken epidermis (Fig. 4). In the latter case the infection thread flattens out to form a suckerlike disk or *appressorium* at the center of which, against the host plant tissue, there forms a peg which, by sheer force or by use of chemical solvents, or by a combination of the two, penetrates into the host tissues. In the case of soil-dwelling, root-infecting fungi, a hypha from the soil-dwelling fungus establishes infection by growing into an injured root or forcing its way into a healthy root.

Once within a host plant, the fungus hypha may grow down between host cells feeding on the intercellular substances. It may produce poisons which diffuse out into the plant tissues, killing the host cells toward which

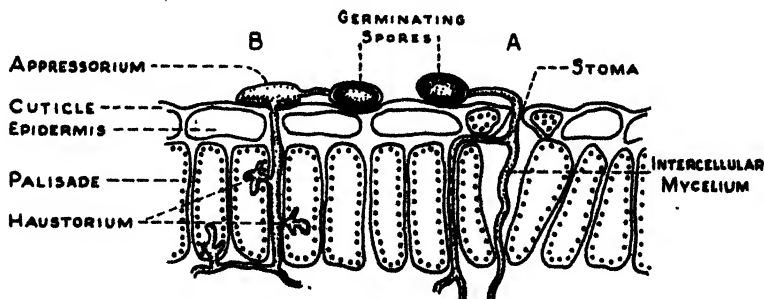


FIG. 4. Infection by fungi. (A) The infection thread has entered the plant through a stoma and formed intercellular mycelium. (B) The infection thread formed an appressorium, forced its way in through the cuticle and developed intercellular mycelium, sending suckerlike haustoria into the cells.

the hypha, by now branched into a mycelial system, grows, feeding on the dead remains of host cells in saprophyte fashion although within a living plant. In the case of other fungi which require living cells for their sustenance, the intercellular hyphae send into the living cells tiny absorbing organs called *haustoria* (singular, *haustorium*) which sap the fluids from the cell until it dies. After a period of feeding on the host plant the hyphae begin to produce spores, on the surface of the plant, singly or in fruiting bodies, or within the plant tissues.

Types of Parasitism. Fungi which require living cells for their nutrition and cannot derive food from lifeless organic matter are called *obligate parasites*. These include some of the most destructive plant disease parasites, such as the rusts and the powdery mildews. Other pathogens spend most of their lives as parasites but if necessary are able to feed on dead organic material. Still other fungi normally are saprophytes, but under unusual conditions can attack and destroy living plant tissues, such as the black bread mold which can be very damaging to harvested sweet potatoes and strawberries or even leaves under very wet conditions.

Among the pathogens are some, such as most of the smuts, which apparently are entirely parasitic in nature, but can be made to grow on lifeless organic matter in laboratory culture. A majority of the fungi alternate between parasitic and saprophytic stages, parasitizing during the growing period of the host plant, then, after the host plant has died, living on its remains as a saprophyte until the following growing season. In such cases it is frequently the nonsexual stage which is parasitic (as the summer stage of apple scab or cereal scab) and the sexual stage which is saprophytic, the sexually-produced spores terminating the saprophytic stage and initiating the parasitic one.

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Chapter 3

Diseases Caused by Basidiomycetes: Rusts

INTRODUCTION TO THE RUSTS

The Basidiomycetes, the order of fungi to which belong the rusts, smuts, fleshy, and woody fungi, are characterized by having spores of the sexual stage (*basidiospores*) typically borne in fours on a club-shaped hypha, the basidium. Apart from this common structure the Basidiomycetes comprise groups of fungi that are very diverse in structure, habits, and pathologic significance.

The Uredinales or rust fungi is a large and highly specialized group, all of which are parasites on plants, in many cases causing diseases of great economic importance. Their high degree of specialization is seen in their narrow choices of host plants and their obligate parasitism.

Rusts can develop only on living cells of their host plants. They have no saprophytic life other than survival as resting spores, and they have resisted all attempts to grow them on laboratory media. Some other types of fungi have broad host ranges and can attack many species of plants, but each species of rust is limited usually to a very few closely related host plant species in a given stage of its life cycle.

The life cycles of rusts may involve up to five different types of spores, each with a different function to perform. All five types of spores may be borne on a single species of host plant (*monoecious rusts*), or more frequently the complete life cycle of the rust includes parasitism of two entirely different host species as little related to each other as wheat and barberry, apple and cedar tree, or pine tree and sunflower (*heteroecious rusts*).

The rust fungi frequently show *physiologic specialization*. By this is meant the existence, within a single species or variety of rust, of numerous *physiologic races* or strains. Normally, the different races of any given rust are identical in appearance and effects on the host plant but they differ physiologically in that each race is able to attack certain varieties of the host species but not other varieties. No two races of a given rust attack exactly the same set of host varieties, and there may be many different physiologic races in a single species or variety of rust. This greatly complicates efforts at rust control by the breeding of rust-resistant crop varieties.

The stem rust of cereals exhibits all of the rust characteristics mentioned above, and this best known of the rusts will serve to illustrate the principles of rust behavior, after which a number of rust diseases of other important crops will be studied.

Rusts of Field Crops

STEM RUST OF GRAINS AND GRASSES (*Puccinia graminis*)

History and Distribution. One of the best known and most important of all plant diseases, stem rust has been a major factor in small grain production for 2,000 years or more. In France in 1660 and in Connecticut (1726) and Massachusetts (1755), long before the relation between barberry and cereal rust was known, shrewd farmers had noticed that grain suffered most severely from the rust when barberries were nearby, and laws were enacted requiring the destruction of barberry near grain fields. De Bary (1864-65) proved the connection of barberry with stem rust.

Stem rust occurs wherever wheat is grown. In North America it is most serious in the northern states and Canada, only rarely causing destructive losses in the southern Great Plains region, in contrast to leaf rust, a distinct disease which always is more injurious than stem rust in the southern wheat areas:

Importance. Stem rust is very dependent on weather conditions, and in some years and sections the crops suffer no damage while at other times the disease has swept north across the Wheat Belt in ruinous epiphytotics. The years 1878, 1904, 1916, 1919, 1920, 1923, 1925, 1935, 1937, and 1938 were marked by disastrous losses from rust. In 1935 stem rust reduced the nation's wheat crop by 160 million bushels—nearly one-fourth of the crop—and certain states, for instance Minnesota and North Dakota, lost 60 per cent of their wheat from rust. The losses are due to low yields,

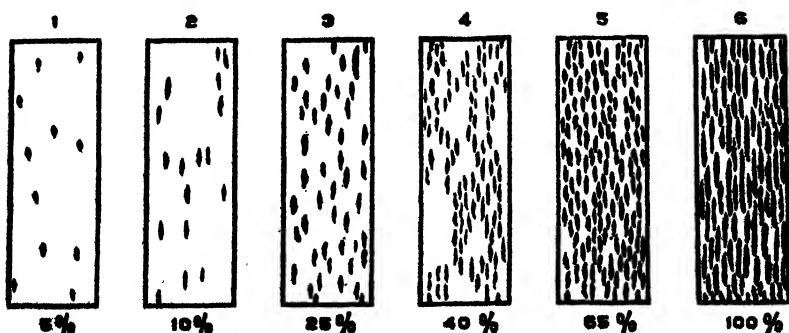


FIG. 5. The modified Cobb scale, in common use for estimating the concentration of cereal rusts on leaves or stems.

poor quality, shriveled grain, and lodging caused by the disease (Fig. 9). Stem rust predisposes plants to winter injury.

The concentration of stem rust on wheat is measured by reference to the official scale given in Fig. 5.

The loss in the crop from stem rust is determined by noting the percentage of infection and stage of maturity of the crop, and reference to Table 1. The figures on rust severity refer to percentages as taken from Fig. 5.

Table 1
RELATION BETWEEN WHEAT RUST SEVERITY AND LOSS IN THE CROP

Stage of Development of the Crop						Loss from Stem Rust
Boot	Flower	Milk	Soft Dough	Hard Dough	Mature	Per Cent
..	(tr)	5	0.0
..	(tr)	(5)	10	0.5
..	..	(tr)	(5)	(10)	25	5.
..	(tr)	(5)	(10)	(25)	40	15.
(tr)	(5)	(10)	(25)	(40)	65	50.
(5)	(10)	(25)	(40)	(65)	100	75.
(10)	(25)	(40)	(65)	(100)	100	100.

Host Plants. *Puccinia graminis* is subdivided into seven varieties: *P. graminis tritici*, principally on wheat, *P. graminis avenae*, principally on oats, *P. graminis secalis*, principally on rye, and others largely on grasses. Each named variety in turn is subdivided into various numbered physiologic races. Thus, *P. graminis tritici* consists of more than 180 physiologic races, each being able to attack certain wheat varieties and unable to attack others. The identity of a rust race is determined by inoculating a group of differential wheat varieties with a rust collection. From the type of reaction produced on each variety the race can be identified by the use of a key. In breeding wheat for rust resistance it is necessary to know the prevalence and distribution of physiologic races in order to provide the wheat with resistance to the races peculiar to the section where the wheat is to be used.

All varieties of stem rust pass to the common barberry and a few other species of *Berberis* for their alternate stage. The Japanese barberry (which has leaves with a smooth outline, in contrast to the spiny-leaved common barberry) is immune from stem rust (Fig. 8).

Symptoms and Signs. On the grain or grass host, stem rust first appears as long, narrow streaks, largely on the stems, but also often on the

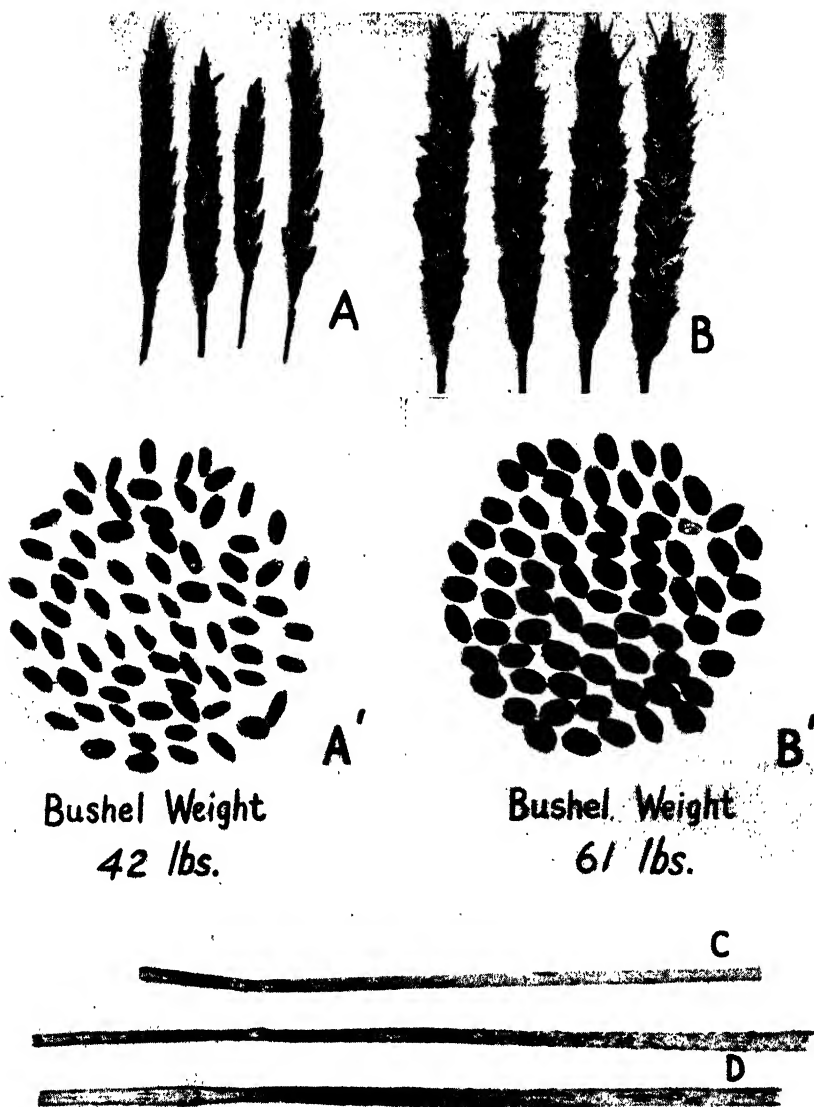


FIG. 6. Stem rust, and its effect on wheat. Note the small heads, shriveled grain, and low test weight of the rusted crop (A, A') in contrast to the healthy crop (B, B'). The latter was secured by sulfur dusting. (C) Showing a stem with an early, uredinial or red stage infection, while the two straws at D are advanced cases in the telial stage. Straws so affected before maturity will result in grain (A, A'). (A, A', B, and B', courtesy F. J. Greaney, Can. Dep. Agr.)







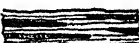
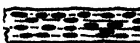




leaf sheaths, leaf bases, or distal part of the leaf blade. It may even occur on the glumes and awns and in rare cases on the grain. The streaks (*uredial sori* or *uredia*) are covered with a dark red powdery mass of one-celled *urediospores*, produced from the feeding mycelium inside the stem or leaf (Fig. 6C). The epidermis is torn back to form a white collar around the sorus. Later the sori become black, as two-celled *teliospores* replace the urediospores in the sori (Fig. 6D). At this stage the stems are often dried and cracked or broken, and the grains are lacking or few in number, shriveled, and light in weight (Fig. 6A, A').

On the barberry the reddish lesions first show on the upper surface of the leaf, dotted with inconspicuous pimples, the *pycnia* (or spermagonia), and soon produce on the under surface of the same lesion a group of white cup-shaped *aecia* (cluster cups), filled with a yellowish, waxy layer of *aeciospores* (Fig. 8). No serious damage to the barberry results from the infection.

There are other rust diseases of cereals which might be confused with stem rust, and these are distinguished as shown in Table 2.

There is confusion in the minds of many growers regarding the rusts of

Table 2
DISTINGUISHING CHARACTERS OF THE LEADING CEREAL RUSTS

	<i>Stem Rust of Small Grains</i>	<i>Leaf Rust of Wheat</i>	<i>Stripe Rust of Wheat and Rye</i>	<i>Crown Rust of Oats</i>
<i>Urediospores:</i>				
Color in mass . . .	Brick red	Bright orange	Yellow	Reddish orange
Shape and relative size				
Appearance of uredium				
Torn epidermis	Conspicuous	Inconspicuous	Inconspicuous	Inconspicuous
<i>Teliospores:</i>				
Shape and relative size				
Appearance of telium	Superficial	Buried	Buried	Buried
Hosts: II-III	Grains and grasses	Wheat (rarely, barley and grasses)	Wheat, rye, grasses	Oats, grasses
0-I	Barberry	Basilisk, in Siberia only	Not known	Buckthorn
Distribution in North America	General	General	Western Canada to Arizona and westward, Texas, Mexico	General

wheat. The two rusts, leaf and stem, are both common and each has a red stage and a black stage. The terms "red rust" and "black rust" may apply to either leaf or stem rust and for this reason the terms should be avoided. When leaf rust is abundant it produces black sori on the leaf sheaths that are often mistaken for the telial sori of stem rust; conversely, the uredial or red stage of stem rust often occurs on the leaves.

Etiology. The life cycle of *Puccinia graminis*, the stem rust organism, is indicated diagrammatically in Fig. 7. About 10 days after a grain plant has become infected, masses of red urediospores appear on the stems or leaves. This stage may be referred to as the "II" stage. The urediospores are the only spores in the rust life cycle that are able to reinfest the same species of plant as that on which they are produced. On this account they are sometimes called "repeating spores." Through this ability to repeat the same type of infection every 10 days, they are the spores that are instrumental in producing rust epiphytotics.

Under favorable weather conditions, the urediospores are liberated and may be carried by the wind from one grain or grass plant to another. In surface moisture (dew or rain) they germinate by sending out a hypha (infection thread) which enters the new host; this establishes a feeding

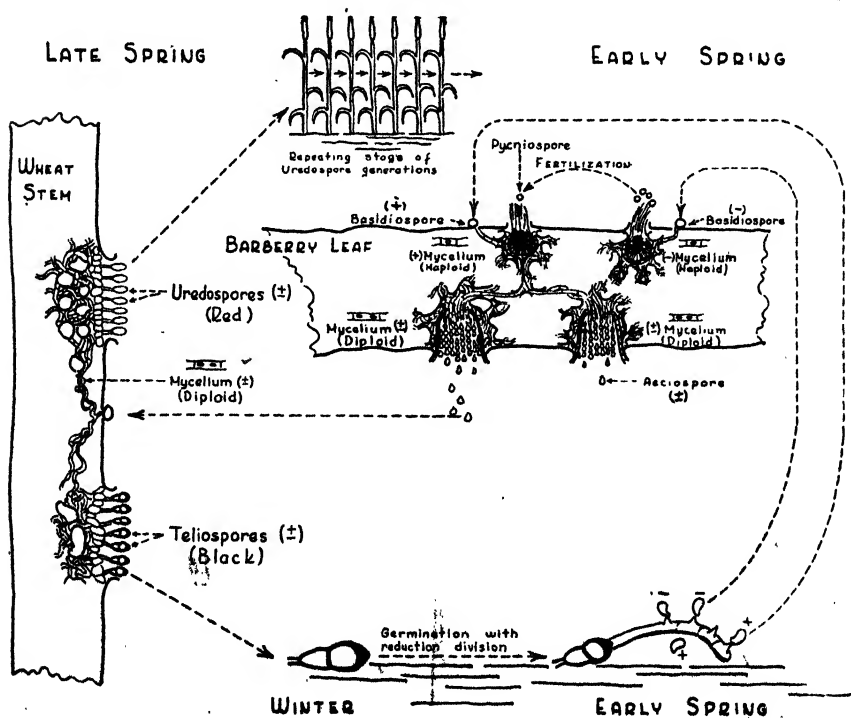


FIG. 7. Diagrammatic outline of the life cycle of stem rust.

mycelium, and produces a new crop of urediospores, all within about 10 days. Many successive generations of urediospores may build up the infestation to epiphytotic proportions. As the grain crop approaches maturity, dark teliospores ("III" stage) begin to appear among the urediospores in the same sori, and soon entirely replace them. Thereafter, one of three things may occur in the case of wheat. In the American wheat areas:

- a. In southern Texas and Mexico the rust lives through the winter, growing on the wheat and producing urediospores. The aecial stage on the barberry has little or no significance. Spring infections are the result of urediospores from the winter infections.
- b. In southern Kansas, Oklahoma, and northern Texas the barberry, which is rarely found infected, plays no part in the spring infection, which is caused by urediospores blown in by south winds from southern Texas and Mexico. The teliospores do not have an active part in the life cycle of the rust under these conditions.
- c. From the northern edge of Kansas to Canada there are two sources of spring infection: (1) the aeciospores from barberry, produced early in the season, and (2) urediospores blown up from the south in ever-increasing numbers later in the season. In the northern states the rust may overwinter as teliospores, which in the early spring germinate and produce basidiospores ("IV" stage), that blow to the barberry and infect it, there producing first pycnia ("O" stage), then aecia with aeciospores ("I" stage). In grain fields that are isolated by mountains, as in Pennsylvania, the barberry is the only source of infection.

In winter wheat areas the land is fallow during the summer, and the fall infections may come from (1) rust that has oversummered in the urediospore stage in grasses or volunteer grain, (2) urediospores blown south from late harvested northern fields or volunteer grain in October and November, or (3) urediospores blown north from summer wheat cultivated in the cool regions of the Mexican mountains.

In oats the life cycle of stem rust appears to be similar to that of the wheat stem rust. The urediospores and mycelium in oats are quickly destroyed by freezing winter temperatures. Spring infection in the southern states is by means of urediospores blown northward from subtropical regions in which the winters are mild enough to permit the rust to overwinter on oats. Farther north, spring infections are due both to urediospores blown in from the more southern states, and to aeciospores from the barberry. There is some evidence that the oat stem rust can oversummer on

grasses as far south as Arkansas, and then pass from the grasses to fall-sown oats.

In barley and rye, stem rust outbreaks are so closely dependent on nearby infected barberry that eradication of the barberry affords a high degree of control of stem rust on these grain crops.

In passing from the grain crop to the barberry and thence back to the grain the fungus passes through a number of important activities. Each cell of the mycelium in the grain plant, each urediospore, and each cell of the young teliospore contains two nuclei. Two sexes occur in the stem rust fungus, designated not as male and female but as $+$ and $-$. Of the two nuclei in a young teliospore, one is of $+$ origin, the other of $-$ origin. As the teliospore matures the $+$ and $-$ nuclei in each cell fuse to form a single nucleus containing both $+$ and $-$ elements. When the teliospore germinates there emerges a four-celled basidium or promycelium and each of the 4 cells then produces a thin-walled, tiny basidiospore. The production of the four-celled basidium is due to two cell divisions of the contents of the spore cell. One of these cell divisions is a reduction division in which the sexual elements become separated again, so that of the basidiospores two are $+$ and two $-$. If the basidiospores chance to fall on a barberry leaf under suitable temperature and moisture conditions, they germinate, send an infection thread into the barberry tissues, develop a feeding mycelium, and after a period of feeding and growth the mycelium grows to the surface and produces flasklike fruiting bodies or pycnia on the upper side of the barberry leaf. Each pycnium contains tiny pycniospores and hyphal threads that extend out through the mouth of the pycnium (receptive hyphae). As each pycnium has developed from a single $+$ or $-$ basidiospore, it and its contents are entirely $+$ or $-$. At this stage sexual union takes place between a pycniospore of one sex and a receptive hypha of the opposite sex. Fertilization is aided by the action of insects. The pycnia also produce a sweetish nectar, and insects, attracted by this nectar, visit one pycnium after another. As they feed they transfer pycniospores much as a honeybee carries pollen from one clover blossom to another and thus fertilizes them. Without this fertilization of the receptive hyphae, little or no further development of the rust is possible.

If a receptive hypha and a pycniospore come into contact, the hypha is fertilized and as a consequence a binucleate or diploid mycelium develops. This more vigorous mycelium feeds on the cells of the barberry leaf, and is massed together on the lower side of the leaf to produce aecia containing aeciospores (Fig. 8). These are unable to reinfect the barberry but if wind-blown to the grain or grass host, they may germinate and infect the grain or grass with mycelium which in a short time gives rise to urediospores.



FIG. 8. Stem rust on barberry; aecia (cluster cups) on the under sides of the leaves. Note the spines on the leaves that distinguish the rust-susceptible common barberry from the smooth-leaved, rust-immune Japanese barberry. (Photograph, U. S. Dep. Agr., Bureau of Entomology and Plant Quarantine.)

The aecial mycelium as well as that in the grain plant is binucleate, and each aeciospore as well as each uredio- and teliospore contains two nuclei, one of each sex. It is not until the maturing of the teliospore that the sexual act occurring in the pycnia finally reaches completion in the fusion of opposite-sexed nuclei to form a single fusion nucleus.

In summary, the rust fungus produces five types of spores, each with a distinct function to perform: (1) The red urediospores spread the rust from grain plant to grain plant. (2) The dark teliospores can infect nothing, but remain on straw or stubble where they may resist winter temperatures, germinating the following spring and producing basidiospores. (3) The basidiospores can carry the rust from the old teliospore on the ground to the barberry. (4) The pycniospores have only the function of sexual fusion. (5) The aeciospores can carry infection from the barberry back to the grain plant.

These different spore types differ in their ability to withstand unfavorable temperatures and other climatic stresses. Most delicate are the

basidiospores, that can carry the disease only for short distances. The urediospores are sufficiently resistant to permit their being carried by the wind for hundreds of miles after which they can settle down and produce infection. Aeciospores are intermediate between these two. The distance to which a spore can be blown without losing its infective ability is important in determining the distance which must separate alternate hosts in order to permit effective rust control.

Epiphytology. The following factors conspire to produce destructive outbreaks of stem rust: (1) large uniform acreages of susceptible wheat varieties; (2) presence of physiologic races of rust that will attack those varieties; (3) mild winters in the south, permitting urediospores to overwinter more abundantly, and farther northward in Texas and possibly Oklahoma; (4) a constant succession of humid, or rainy days and dewy nights as the crop matures in the spring, providing the required conditions for urediospore germination; (5) cool temperatures (with an optimum in the vicinity of 65° to 75°F.); (6) rank, succulent growth of the crop (in general, the rusts and other obligate parasites are most destructive on the most vigorous, succulent plants, especially those in well-watered soils which are high in nitrogen); (7) late maturing crops which have longer exposure to the rust; (8) continuous south winds; and (9) in the northern areas, presence of barberry. The greatest losses result when these factors are combined with hot, dry conditions just before harvest, which increases the suffering of the plants already deprived of water by the inadequate functioning of the stems. Epiphytotics of stem rust are associated with a combination of most or all of the factors listed.

Control: Prevention of stem rust depends on several practices, all of which contribute to reductions in the losses from the disease:

1. **RESISTANT VARIETIES.** Much effort has been expended in attempts to breed wheat and other grains resistant to stem rust. This work has largely centered in the northern states where stem rust losses are greatest. The history of rust resistance in wheat shows some of the difficulties encountered.

Kanred wheat was a selection from a Russian wheat brought to America in 1906. In 1916, when it was being grown in Kansas, it was discovered to be stem rust-resistant. It was distributed, and by 1924 over 4,000,000 acres were planted with this variety in Kansas and adjacent states. As years passed, however, it began to lose its rust resistance, and became attacked more and more severely by stem rust. This weakness, together with a tendency to lodging, was such that by 1929 its acreage had dropped 20 per cent, and this drop has continued since. Kanred lost its resistance, not because of any change in the wheat, but because new physiologic races of the stem rust fungus had appeared, races that were able to attack Kanred.

Meanwhile another wheat, Ceres, was coming to the fore as a stem rust-resistant wheat for northern areas. First distributed in 1926, by 1933 it occupied 5,000,000 acres in the United States and Canada, and by 1935 this acreage had increased still further. At the same time, an obscure race of stem rust, race 56, had been rapidly increasing, a race that could attack Ceres. With the enormous acreage of susceptible Ceres wheat and favorable weather conditions in 1935, race 56 of stem rust swept across the northern plains in the greatest rust epiphytotic of history, and destroyed one-fourth of the nation's bread grain. To replace Ceres, the rust-resistant Minnesota variety, Thatcher, introduced in 1934, soon occupied the great acreages formerly devoted to Ceres. Thatcher, being resistant to race 56 and other common stem rust races, has shown little stem rust in the field, but is highly susceptible to leaf rust, and suffered severely during the 1938 epiphytotic. The variety Newthatch, bred for resistance to both rusts, was soon released to replace Thatcher but it, too, developed susceptibility to leaf rust, evidently because of a change in leaf rust races. Other spring wheats that have been bred for stem rust resistance include Cadet, Coronation, Henry, Mercury, Merit, Mida, Pilot, Redman, Regent, Renown, Rival, and Vesta, all but the last being moderately to highly resistant to leaf rust as well. Carleton and Stewart are among the newer durum wheats possessing resistance to both rusts.

Most of the commercial winter wheats are susceptible to one or both rusts, but a few, including Austin, Blackhawk, Chiefkan, Comanche, Illinois No. 2, Iobred 73, Iowin, Kawvale, Marmin, Nebred, and Prairie, are intermediate or resistant in reaction toward stem rust. Some early maturing varieties, such as Early Blackhull, while susceptible to rusts, escape their worst effects by maturing before rust becomes most abundant.

The production of rust-resistant wheat varieties remains our most important means of fighting stem rust, but the histories of Kanred and Ceres show us that the breeding work is never finished, that new resistant varieties will constantly be needed, as new physiologic races of the rust become prevalent, and that the best efforts of the wheat breeder will succeed in keeping only a few years ahead of the rust.

The widespread adoption of stem rust-resistant wheat varieties in the "rust area" of Canada has been shown to have increased Canadian wheat production by 41,339,000 bu. per year, valued at \$27,242,000. The profit in using these disease-resistant varieties in a single year has repaid 13 times over the total expenditure made by Canada for wheat rust research through the years. In South Dakota, during the rust epiphytotic of 1944, the resistant varieties Pilot and Rival outyielded the susceptible Marquis by as much as 800 per cent; without rust resistant varieties the South Dakota wheat

crop of that year would have been almost a total failure instead of one that was almost double the preceding ten-year average.

2. **ERADICATION OF BARBERRY.** In 1918 the United States Department of Agriculture, in coöperation with 13 of the central states, began a campaign to eradicate the common barberry over this great area. By 1945 the eradication area extended from Washington and Colorado to Pennsylvania and Virginia, and over 100,000,000 bushes had been destroyed at a cost of nearly \$3,000,000 per year (Fig. 209). In Minnesota, bounties of \$2.00 to \$5.00 have been paid for each barberry bush found and shown to eradication officials. We now know that eradication of the barberry will not eliminate the disease because of infection by urediospores blown up from the South. There are some who feel that the eradication program has been wasted effort. But even though barberry eradication will not wipe out stem rust, there remain three good reasons for continuing the work:

- a. We have seen that the efforts of the plant breeder often are thwarted by the new physiologic races of rust. In many cases these races are the result of hybridization of older races during sexual reproduction of the rust which takes place *on the barberry*. Eradication of the barberry aids by destroying the breeding place for new rust races.
- b. The damage from stem rust depends greatly on the stage of maturity of the wheat when it is attacked. A 10 per cent rust attack (see Table 1) when the wheat is in the boot stage will result in 100 per cent loss by harvest time, while a 10 per cent attack in the hard dough stage will produce only a 5 per cent loss in the crop. The barberry brings infection to northern wheat fields much earlier than it would arrive via the south winds, hence greatly increases the rust damage.
- c. In sheltered wheat sections, protected by mountains, west of the Rockies or east of the Appalachians, no urediospores blow in from the outside, and the only infection of wheat is from the barberry. In these sections barberry eradication gives 100 per cent control of the disease. The barberry is especially important in initiating local epiphytotics in rye and barley.

3. **AGRONOMIC PRACTICES** that diminish losses from stem rust include avoidance of low, poorly-drained sites for wheat, avoidance of excessive rates of planting or excessive nitrogen fertilization, and the use of early maturing varieties.

4. **SULFUR DUSTING.** It has been proved for a number of years that applications of sulfur dust will control rusts (Fig. 9). Early views that this was impractical are being reconsidered in view of the recent development

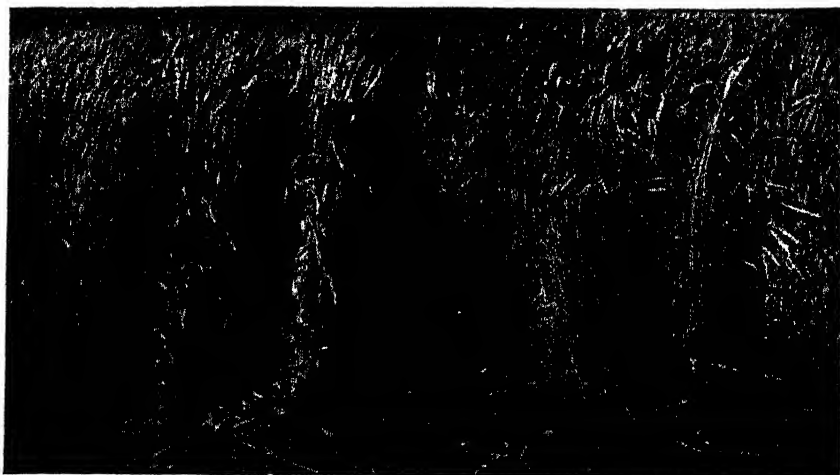


FIG. 9. The destructiveness of rust in wheat. In the rusted rows at the right note the weak, broken straw, and paucity of well-filled heads, in contrast to the rust-free plants at the left. In this case the greater part of the damage was due to stem rust; much less to leaf rust. The healthy plants are of the same variety as the rusted ones, but have been protected by dusting with sulfur. (Courtesy, F. J. Greaney, Can. Dep. Agr.)

of airplane dusting methods (Fig. 223). Experiments in Canada, New York, Minnesota, and Australia have shown clearly that under conditions of heavy rust infestation the sulfur treatment has more than paid for itself in increased yields. It should be remembered, too, that rust is most abundant in the years of ample rainfall when potential yields are high and when the cost of the dust treatment represents a proportionately small part of the potential return. Farmers have been reluctant to undertake this treatment without more information on profit and cost over a period of years, as well as because of the relatively small margin of profit. At present the experimental evidence is not extensive enough to justify recommending dusting as a general practice, but it is a desirable control measure in growing small plots of valuable grain for seed production or for show purposes.

LEAF RUST OF WHEAT (*Puccinia triticina*)

This rust disease, which occurs wherever wheat is grown, is more serious than stem rust in the southern half of the American wheat belt. Each year it causes losses of a few per cent in the American crop, and occasionally it breaks out in epiphytotic force, as in 1938 when it caused losses up to 30 per cent in several states from Texas to Canada. In many individual fields the loss was complete. Where stem rust quickly destroys the crop by cutting off the water supply, leaf rust progressively destroys the

leaf tissue throughout the season, resulting in a reduced number of kernels, shrivelled grain, low test weight, and low protein content. It causes wheat plants to transpire and respire at greatly increased rates, which reduces their food-storing capacity and wastes soil moisture, predisposing the plants to drought injury.

Host Plants. *Puccinia triticina* attacks wheat and a small number of grass species. On the latter, however, the rust is not highly infective and these play little part in the cycle of the rust. There are a number of closely related leaf rusts of other grasses, but these will not attack wheat. *P. triticina* consists of many physiologic races, each restricted to certain wheat varieties and distinguished by their reactions on five differential wheat varieties.

In Siberia there is an alternate O-I stage on the basilisk (*Isopyrum* species), which is necessary for the perpetuation of the rust, but no alternate host has been found naturally infected in any other part of the world. The meadow rue (*Thalictrum* species) has been infected experimentally with the O-I stage of leaf rust but has not been found infected in nature.

Symptoms and Signs. (Refer to Table 2 for a comparison with stem rust.) Wheat leaves and leaf sheaths become covered with small, round or oval, bright orange uredial pustules (Fig. 10). The disease begins in the lower leaves and works toward the top of the plant. Soon after attack the leaves die progressively upward until in severe early attacks every leaf may be destroyed before the heads emerge from the boot. Later, the leaves and sheaths display short, dark, lead-gray telial pustules (Fig. 10). These are buried under the epidermis and the teliospores are not in exposed masses as in stem rust. In heavy infestations the urediospores become so abundant that one's clothes are reddened with them, and the infested field is discolored reddish-brown or yellow when seen from a distance. The kernels may entangle so many urediospores in the brush as to give the seed a red discoloration sometimes puzzling to farmers or millers.

Etiology. Under American conditions the urediospore is the only effective spore form. The alternate host is never found infected, hence the teliospores play no part in the cycle of the rust. The urediospores are able to endure low temperatures, and the fungus overwinters as mycelium in winter wheat, sometimes as far north as Canada, but principally in the southern part of the wheat belt, actively producing successive crops of urediospores. Under favorable conditions these may rapidly increase in 10-day cycles in the spring. Although the urediospores are cold-resistant they are unable to endure high summer temperatures, and die out during hot summers in southern regions. In such cases the fungus follows the crop northward during the summer and returns to the fall-grown wheat

in the south via the north winds of October and November. In exceptionally mild summers the fungus appears to be able to oversummer on volunteer wheat even in the South. Under suitable conditions the infestation of fall wheat may become so abundant as to injure the value of the wheat for pasture, followed by poor winter survival of the crop.

The complete life cycle, as it occurs only in the Lake Baikal area of Siberia, resembles that of stem rust. The teliospores produce basidiospores or sporidia in the fall or spring; these are borne to the little biennial wheat field weed, the basilisk (*Isopyrum*), and infect it, the mycelium first producing pycnia on the upper surface of the leaf, and later aecia on the under-surface, the aeciospores carrying the infection back to the wheat. This is the only method of winter survival in the rigorous climate of east-central Siberia where the rust cannot overwinter in the uredio stage nor be blown in from other regions.

Epiphytology. The leaf rust fungus differs from stem rust in being adapted to cooler temperatures and having better ability to survive winter



FIG. 10. Wheat leaf rust. (*Left*) Seedling leaves showing uredial pustules in an early stage of infection from artificial inoculation. (*Right*) Telial stage on older leaves which have been killed by the fungus. The teliospores are buried under the leaf epidermis.

cold. Epiphytotic development follows extended periods of damp weather with temperatures in the vicinity of 50° to 65°F. Above 80°F. infection is erratic or does not occur. High temperatures after infection during the incubation period result in masked or symptomless infection. The urediospores are somewhat more resistant to cold and less resistant to heat than in the case of stem rust.

Control: 1. RUST RESISTANT VARIETIES. Less has been done in breeding wheat for leaf rust resistance, than with stem rust although work in this direction is proceeding in various state agricultural experiment stations. With the exception of Vesta, all the varieties of spring wheat with resistance to stem rust listed on p. 32 are resistant also to leaf rust. Of the hard red winter wheats, the recently released varieties, Austin and Westar, are quite resistant to leaf rust, and moderate resistance is found in Pawnee, Kawvale (semihard), and Reliant. Some formerly resistant varieties, such as Tenmarq and Mediterranean, are now listed as susceptible. Leaf rust-resistant soft red winter wheats include Prairie, Sanford, and Wabash. Durum wheats as a class are resistant to leaf rust. Blackhawk, a hardy white winter wheat released by Wisconsin breeders in 1945, combines good leaf rust resistance with resistance to bunt and is less susceptible than many other commercial wheats toward loose smut and stem rust. The introduction of Sanford wheat in Georgia is estimated to have increased the annual value of the wheat crop in that State by \$400,000. There are numerous highly resistant noncommercial wheats which may be used as breeding parents for developing rust-resistant commercial varieties for the future.

2. SULFUR DUSTING. (Refer to discussion under stem rust.) The two rusts, leaf and stem, often develop together as their environmental requirements are similar. Sulfur dusting would have double value in such a case. As in the case of stem rust, no general recommendation can be made, but dusting is advised in rust years on small plots of valuable seed or show grain.

3. AGRONOMIC PRACTICES favoring rust control consist in avoiding low, undrained sites for wheat, the use of early varieties, practices which encourage earliness in the crop, and avoidance of excessive nitrogen in the soil.

4. BIOLOGICAL CONTROL. Leaf rust pustules frequently are parasitized by the fungus, *Darluca filum*, a parasite on rust spores and mycelium. Thus far it has not been possible to make practical use of *Darluca* in rust control, but in nature it undoubtedly plays a part in keeping the rust fungi in check.

STRIPE RUST OF WHEAT AND OTHER GRAMINEAE (*Puccinia glumarum*)

Stripe rust has been known since 1892 in the northwest quarter of the United States and adjacent Canada. It has been found also at higher altitudes in Mexico, at various points in California, and in Arizona. In 1941 it was found to be abundant in the cereal nurseries of the experiment station at College Station, Texas. No serious spread of the disease resulted, probably because the Great Plains do not provide the temperature requirements of the rust. Stripe rust is regarded as one of the most important of the cereal rusts in Northern Europe and China.

Stripe rust can be distinguished from the other cereal rusts in that the uredial stage is yellow, and the pustules occur in streaklike clusters on the leaves. Later, a telial stage develops in black streaks. Glumes, necks, leaf sheaths, and even kernels are attacked, resulting in poor yields and shriveled grain of poor viability.

Wheat is the main host, although barley and rye are attacked as well as some 60 species of grasses. There are two varieties of the rust, one primarily on wheat, the other mainly on barley, with more than 50 physiologic races.

The O-I host of stripe rust is unknown and its perpetuation is through urediospores. Survival of the rust between crops appears to be on volunteer or planted wheat or barley at cool latitudes and altitudes. Epiphytotics are favored by abundant late summer infection, winter survival, and cool spring weather with abundant moisture and sunshine. The urediospores germinate best at 53°F. and quite poorly above 68°. This is one reason why the disease is checked by hot weather.

Stripe rust can be controlled by resistant varieties of which the leading ones are:

Soft red winter wheats: Clarkan, Denton, Forward, Fulhio, Kawvale, Leap, Mediterranean, Minhardi, Nittany, Penquite, Red Chief, Redhart, Red Rock, Red Russian, and Trumbull.

Hard red winter wheats: Blackhull, Cheyenne, Eagle Chief, Early Blackhull, Iowin, Kanred, Kharkov, Minturki, Oro, Purkov, Ridit, Turkey, and Wisconsin Pedigree No. 2. Tenmarq varies from susceptible to resistant.

Spring wheats: Defiance, Dicklow, Garnet, Haynes Bluestem, and Thatcher.

CROWN RUST OF OATS (*Puccinia coronata*)

This common leaf rust of oats with rounded, light orange uredial sori and buried telial sori, is often very destructive, the losses paralleling those from leaf rust in wheat. The alternate host is the buckthorn (*Rhamnus*

species) which is important in the epiphytology of the rust from Iowa northward. Farther south crown rust survives the winter as mycelium in the oat plant. The rust is distinguished by the teliospores which are surmounted by a circle of little projections, as in a king's crown. The disease is controlled mainly by the use of resistant varieties.

Crown rust-resistant varieties of the past, such as the Red Rustproof group and Bond, have been showing increasing rust susceptibility owing to the increase in physiologic races of rust that can attack these varieties. In their place have been developed many new varieties with crown rust resistance often combined with resistance to stem rust and smut among other valuable qualities. The Victoria oat, an unadapted and late but vigorous variety with resistance to most crown rust races and to smut, has been a parent of many of these newer varieties, which include Boone, Marion, Control, Tama, Neosho, Ventura, Vicland, Cedar, Vikota, Clinton, Benton, Bonda, Mindo, Eaton, and Forvic for the northern half of the United States, and Neosho, Osage, Victorgrain, Fultex, Ranger, Rustier Quincy, Camellia, Traveller, and Florilee for the southern area. Recent destructiveness of the new Victoria blight on oats of Victoria parentage (p. 180) limits the usefulness of these varieties for the future.

CORN RUST (*Puccinia sorghi*)

With the O-I stage on sorrel (*Oxalis*), corn rust is common but rarely damaging enough to warrant control measures.

BARLEY LEAF RUST (*Puccinia anomala*)

This has an O-I stage on the star-of-Bethlehem (*Ornithogalum* spp.) in Europe, but in America dispenses with this stage. It is one of several barley leaf diseases that can be at times quite destructive. In Texas, where this disease is a limiting factor in barley production, the new rust-resistant variety Tunis offers a solution, and there are numerous other barley varieties with resistance to leaf rust.

RYE LEAF RUST (*Puccinia dispersa*)

With its O-I stage on *Anchusa*, and habits much like the leaf rust of wheat, rye leaf rust is often very common, but because of the earliness of maturity of the rye crop, its leaf rust is not usually regarded as a serious problem.

SORGHUM LEAF RUST (*Puccinia purpurea*)

This has no known O-I stage. Like corn rust it is rarely severe enough to justify efforts at control, but if this is needed, numerous sorghum varieties are resistant.

FLAX RUST (*Melampsora lini*)

This offers a serious problem in flax culture wherever flax is grown. In 1942, for example, it reduced the North Dakota flaxseed crop by 25 per cent or 2,000,000 bu. It has a complete life cycle with all five spore stages occurring on flax and occurs in at least 14 physiologic races. Control is aided by rotation with other crops and removal or plowing under of flax refuse, but chiefly depends on the use of rust-resistant varieties such as Ottawa, Calar, Viking, Norsk, Arrow, Golden, Sheyenne, Renew, Royal, Walsh, and Rio.

SUNFLOWER RUST (*Puccinia helianthi*)

This rust is very frequently found on wild sunflowers, and when this plant is grown commercially, for the production of oil as in Russia, the rust can become a limiting factor to the success of the crop. So important is this that a program of breeding sunflowers for rust resistance is under way in Russia.

ALFALFA RUST (*Uromyces striatus*)

Alfalfa rust is a common alfalfa leaf disease, causing premature defoliation and sometimes seriously reducing hay and seed production. The aecial stage is on *Euphorbia* but is poorly understood in America where it appears to have little importance, the rust evidently overwintering on the alfalfa. Frequent mowing will save some of the rusted foliage, and certain selections of alfalfa show high resistance to rust and will serve as a basis for control in the future.

CLOVER RUSTS (*Uromyces elegans* and other species)

Clover rusts are common and often quite injurious. All known spore stages occur on the clovers. There is considerable resistance seen in individual plants and clover varieties, offering the basis for control by selection. Early harvesting of clover hay and the burning of fields in early spring are sometimes useful as control measures. In the case of *U. elegans* on Carolina clover an increase of 50 per cent in hay tonnage has been reported following dusting with sulfur.

Rusts of Grasses

A number of grass rusts are very common and often injurious to pasture during seasons favoring the cereal rusts (Fig. 11). Little can be offered for their control. At times, Bermuda grass suffers seriously from rust and when this grass is used for lawns the disease could be kept under control by reduced watering and by dusting with sulfur. Little barley

(*Hordeum pusillum*) may be heavily rusted under conditions favoring wheat leaf rust so that its forage value is considerably decreased. Numerous wild plants serve as the aecial hosts of the grass rusts, including members of the crowfoot, milkweed, honeysuckle, goosefoot, and primrose families and a number of others.

Rusts of Trees and Shrubs

CEDAR APPLE RUST (*Gymnosporangium juniperi-virginianae*)

This important rust is most destructive to apple (O-I stage) but at times is a nuisance in ornamental cedars (*Juniperus* species) which support the telial stage. The urediospore stage is omitted from the life cycle. Since there are no repeating spores (urediospores) on either host, the disease can persist only where both hosts are present, and may be controlled by eradication of either cedar or apple. Two very similar species are *G. globosum* which passes from cedars to the hawthorn, and *G. germinale* (= *G. clavipes*) which affects cedars and quince trees.

In infected apple the leaves show abundant reddish lesions, stippled with black pycnia on the upper surface and with long, funnel-shaped aecial cups on the under side (Fig. 12). The walls of the aecium split at several points and roll back, giving the sorus a starlike appearance. Affected trees show serious reduction in the amount and quality of the harvested crop.

The aeciospores blow to the cedar in the summer and infect the young twigs. The infection is not obvious until the second spring after, when the lesions are seen as round galls on the twigs, $\frac{1}{2}$ inch or larger in diameter. In moist weather these galls develop long, orange, jellylike horns which consist of masses of teliospores with long gelatinous stalks (Fig. 12). The teliospores germinate at once, producing basidiospores, which are carried by the wind to nearby apple trees and bring about the leaf infections. The fungus is tolerant of temperature within wide limits, but requires moisture for infection. It does not develop in epiphytotic proportions since there are no repeating spores.

Control. Control is most readily accomplished by removing either



FIG. 11. This rust, *Puccinia peridermio-spora*, on tall marsh grass, *Spartina pectinata*, is representative of the many rusts that affect our native grasses, sometimes being quite injurious to pasture.



FIG. 12. Cedar-apple rust. (*Top, left*) Pycnial stage on upper surface of apple leaf. (*Top, right*) Aecial stage of the closely related hawthorn rust on hawthorn twig, showing swellings and distortion. (*Bottom, left*) Aecial stage on apple fruit. (Courtesy, N. Y. Agr. Exp. Sta.) (*Bottom, right*) Telial stage on red cedar.

host from the vicinity of the other. Twelve states have laws requiring the eradication of cedars from apple districts, the standard requirement being a cedar-free zone a mile wide around orchards. Such a wide zone is not necessary for a practical control, since the basidiospores are so widely

dispersed, even at a distance of a few hundred yards from cedars, as to remove the danger for practical purposes. Noncommercial apple trees can be removed to protect ornamental or nursery cedars. For apple rust control by spraying the standard material is Fermate, $\frac{1}{2}$ to $1\frac{1}{2}$ lbs. in 100 gal. to which may be added 3 lbs. of wettable sulfur or 6 lbs. of flotation sulfur paste for scab control, as prepink, full pink, and calyx sprays. A rust-resistant seedling of the common red cedar has recently been discovered in West Virginia, and the native southwestern species *Juniperus mexicana* appears to be immune from apple rust. Wherever they are adapted, either of these could be substituted for the common cedar in ornamental plantings near orchards.

BLISTER RUST OF FIVE-NEEDLE PINES (*Cronartium ribicola*)

The blister rust fungus came to the United States from Europe about 1900. Since then it has caused enormous losses from New England through the Lake States, and into the virgin timber of the Pacific Northwest. The pycnial and aecial stages occur on the pine, and the uredial and telial stages on currants and gooseberries of the genus *Ribes*. While blister rust has a limited distribution, it is of special interest because of the extensive federal eradication project which resulted in the extermination of *Ribes* from over 6,000,000 acres in a 10-year period.

The fungus is perennial in the pine, and once a tree is infected, the mycelium gradually spreads until the tree is destroyed. Affected trees show dead branches ("flags"), slight swelling of the bark, tiny yellow droplets of ooze from the pycnia, and white, thin-walled blisters, the aecia, filled with yellow aeciospores. Bleeding of resin is common. The aeciospores carry the fungus to the currants or gooseberries, and infect the leaves, producing orange-yellow uredial pustules and columns of teliospores on the leaf lesions. This is a serious disease on the currant and gooseberry bushes as well as on the pines (Fig. 13).

Large amounts of money have been spent on the government control program of *Ribes* eradication, but the program has been successful in protecting the eradicated areas. Since there is no repeating spore on the pine, the *Ribes* need be removed no more than 1000 feet from pine in order that the pine be protected. Eradication has been by means of fire, by hand, by chemicals, and by a system of forest management which does not favor the establishment of *Ribes*. Eradication is a local problem, and its practical value varies from one locality to another according to the type and distribution of the *Ribes*, and the abundance and value of the pines. The problem of eradication in the Pacific Northwest is the most serious one at present because the infested areas are remote from settlements and

inaccessible, and because huge areas are involved if the valuable pines of the west coast are to be saved.

Control of blister rust also involves the use of resistant varieties of pine and *Ribes*. Several commercial varieties of currants, including Viking and Red Dutch, have been found to have a satisfactory degree of resistance. In Wisconsin, resistant pine selections have been made, and a method developed for their rapid propagation by use of cuttings treated with growth hormones.

There are many other coniferous rusts with the aecial stage producing white, columnar sori on the needles. A common example is the needle rust of pines, *Coleosporium solidaginis* with the II-III stage on many species of asters. While these ordinarily are unimportant on either host, they are occasionally destructive in coniferous nurseries. Removal of the alternate host, usually a weed, is the preferred means of control.

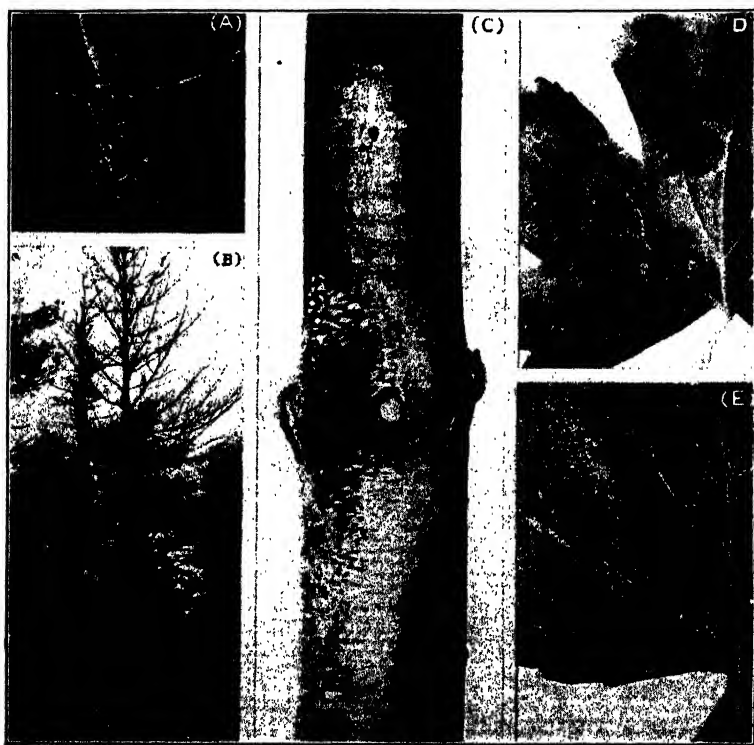


FIG. 13. Blister rust of pines. (A) Young branch canker showing typical spindle-shaped swelling. (B) Merchantable pines killed by the rust. (C) Trunk canker showing aecia, pycnial scars, and region of bark discoloration. (D) Uredial stage showing pustules on under side of *Ribes* leaf. (E) Telial stage showing columnal development on under side of *Ribes* leaf. (Courtesy, New England Sec., Soc. Am. Foresters, Leaflet 26.)

RUST OF STONE FRUITS (*Tranzschelia pruni-spinosae*)

The rust of stone fruits is common on plums and related fruits and is often quite harmful in the latter part of the growing season, the leaves being covered with extensive brown uredial and telial spore masses. The alternate hosts are members of the crowfoot family: anemone, hepatica, buttercups, and meadow rue. Fungicidal spraying affords the best means of control.

ORANGE RUST OF CANE FRUITS

(*Gymnoconia interstitialis*)

This rust is a familiar and very conspicuous disease of black and purple raspberries, blackberries, and dewberries, the leaves becoming covered with brilliant orange aecial spore masses. The rust has an unusual life history. The orange aeciospores blow to raspberry leaves and cause local infections resulting in the production of brown telial sori. On germination of the overwintered teliospores in the spring the basidiospores infect the buds at the tips of the canes and this infection becomes systemic, the rust mycelium invading the entire plant and then producing great numbers of orange aeciospores in midsummer. The plants become practically worthless. Owing to the systemic nature of the disease, control measures include propagation from rust-free plantations and, in cases of light infestation, cutting out and burning the rusted canes. Superficial examination of incoming plants of unknown source is of little value because the mycelium within infected stocks cannot be seen except by careful microscopic examination.



FIG. 14. Asparagus rust. Telial pustules on asparagus stem, enlarged about 3x.

Rusts of Vegetables

ASPARAGUS RUST (*Puccinia asparagi*)

At one time asparagus rust was a serious menace to asparagus culture in America, and almost eliminated this crop in important areas. Since then it

has been so generally controlled by planting of the popular rust-resistant varieties Mary Washington and Martha Washington that it is no longer a major problem. Recently, in some localities, an increasing amount of rust has been observed on the Washington varieties which growers take to indicate that these varieties are losing their resistance. This could be explained by a change in the variety, due to cross pollination, or to a new and more virulent race of the asparagus rust fungus. All spore stages occur on asparagus leaves and stems. The telial stage is illustrated in Fig. 14.

BEAN RUST (*Uromyces phaseoli*)

This rust occurs on common and lima beans wherever these crops are

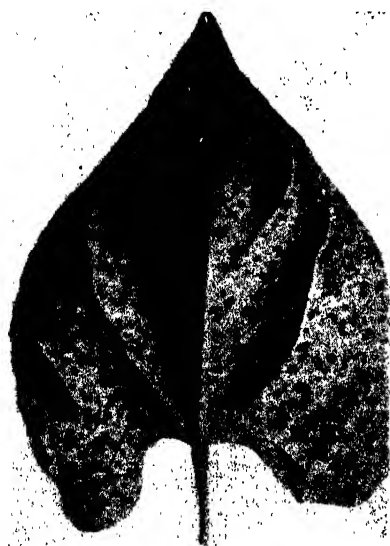


FIG. 15. Bean rust. (Courtesy, S. A. Wingard, Va. Agr. Exp. Sta.)

grown. All spore stages occur on the leguminous host (Fig. 15). The disease, which can be very destructive, is controlled by dusting or spraying the vines with a sulfur fungicide or by the use of rust-resistant varieties. The use of sulfur has nearly doubled bean yields in Florida, Colorado, and Oregon. The green snap bean, Wisconsin Refugee, Ala. No. 1, and certain selections of pole beans of the Kentucky Wonder type are resistant to some of the races of bean rust. The true bean rust should be distinguished from bean anthracnose (p. 194) and bacterial blight of beans (p. 270) which are often loosely called "rust" but are not caused by rust fungi. "Rust resistant" beans

listed in seed catalogs are likely to be anthracnose-resistant but susceptible to the true rust.

SWEET POTATO RUST (*Coleosporium ipomoeae*)

This rust is rare in commercial sweet potato plantings except in the tropics but occurs commonly on the wild sweet potato *Ipomoea hederacea*. The O-I stage on pine trees is sometimes quite destructive in southern nurseries, in which case eradication of the weed host is suggested.

Rusts of Ornamentals

CARNATION RUST (*Uromyces caryophyllinus*)

One of the commonest diseases of carnations, especially in the greenhouse, is carnation rust. Typical brown powdery lesions disfigure the leaves (Fig. 16C). Control is accomplished by avoidance of syringing (often used for red spider control), dusting with sulfur before blooming time or spraying with Fermate or Dithane, maintaining relatively dry air in the greenhouse, and taking cuttings only from rust-free plants.

SNAPDRAGON RUST (*Puccinia antirrhini*)

Dark brown powdery sori on all green parts of the host are caused by the common snapdragon rust. (Fig. 16A.) It often requires control in the greenhouse, the methods being those used for carnation rust with the addition that numerous rust-resistant snapdragon varieties are available. Maintenance of greenhouse temperature near 70°F. and propagation of



FIG. 16. Rusts of ornamental plants. (A) Snapdragon rust. (B) Hollyhock rust. (C) Carnation rust. (A, courtesy P. E. Tilford, Ohio Agr. Exp. Sta.; B and C, courtesy P. P. Pirone, N. J. Agr. Exp. Sta.)

snapdragons by seed instead of by cuttings will reduce the snapdragon rust problem.

HOLLYHOCK RUST (*Puccinia malvacearum*)

Hollyhock rust is exceedingly common, the undersurfaces of leaves often being almost wholly covered with tan to purplish telial sori (Fig. 16B). The rust is unusual in that all spore stages are lacking except the teliospores and basidiospores. Control depends on fall burning of the old hollyhock and mallow debris, removal of the first rusted leaves, and sulfur dusting in the spring.

OTHER RUSTS OF ORNAMENTALS

Many other ornamental species suffer from rust diseases and because the value of ornamentals depends on their appearance, rust attacks too light to devitalize the plant may still impair the plant's esthetic value. The commoner ornamentals with rust diseases include roses, violets, clematis, phlox, morning glory, sunflowers, pansies, and verbenas. Control in nearly all cases is a matter of fall sanitation and preventive applications of standard fungicides in the spring.

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Chapter 4

Diseases Caused by Basidiomycetes: Smuts

Nature of the Smuts

Like the rusts, the smuts are basidiomycetes, but their life histories are much simpler than in the rusts. Only two types of spores are concerned, the *chlamydospores* which are usually in abundant black masses, and the *basidiospores* or *sporidia* which are produced by the chlamydospores much as in the case of germinating rust teliospores. Chlamydospores are transformed vegetative cells of fungus mycelium which become rounded and thickwalled, and are often quite resistant to aging and able to survive periods between crops, among seed or in soil. In germinating, the smut chlamydospores produce a thread- or clublike *promycelium* which bears four or more sporidia, either at the sides of the promycelium, as in the genus *Ustilago*, or at its tip. (Fig. 17.) There are three genera of smuts with germination of the latter type, *Tilletia* with simple, dusty chlamydospores, *Entyloma* with simple chlamydospores buried in the host tissues, and *Urocystis* in which the dusty chlamydospores each consist of one or more central fertile cells ordinarily surrounded by a coat of sterile, protective cells.

The smuts are primarily a grain- and grass-inhabiting group, although there are a few exceptions, such as the onion smut and the white smuts of dahlia, water lily and a few other dicotyledons. In nature the smuts appear

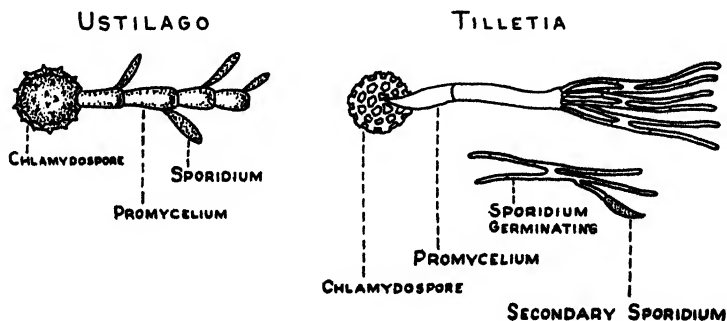


FIG. 17. Chlamydospore germination in two leading genera of smuts. (*Ustilago*, after Smith. *Tilletia*, after Heald.)

to live as obligate parasites, developing only on the living host plant though they may be grown experimentally on nutrient media.

Control of the smuts depends on their life histories. There are three distinct types of life history to be found among them, as indicated in Table 3. In one of these (e.g. corn smut) the infections are local, occurring on various above-ground parts of the plant, each spore mass (as in most rust diseases) resulting from a separate infection. Other smuts produce systemic infection: from a single inoculation the fungus mycelium spreads throughout the plant, unseen until the flowering or fruiting, when the reproductive structures are found to be transformed into smut spore masses.

Table 3
CHARACTERISTICS OF THE THREE TYPES OF SMUT DISEASES

	Seedling Infection Type	Blossom Infection Type	Local Infection Type
Place of primary infection	Seedling	Blossom	Leaves, etc.
Nature of infection.....	Systemic	Systemic	Local
Transmitted from crop to crop.....	On the seed	In the seed	In the soil
Principal control measures	Seed dusting with fungicides	Hot water seed treatment	Sanitation, rotation
Examples.....	Bunt of wheat Covered smut of oats Loose smut of oats Covered smut of barley Black loose smut of barley Covered smut of sorghums Millet smut Stem smut of rye Flag smut of wheat Kernel smut of rice	Loose smut of wheat Brown loose smut of barley	Corn smut Head smut of corn and sorghums White smuts of spinach and ornamentals Onion smut Leaf smut of rice

Survival of the smuts is by means of chlamydospores on the seed or in the soil, or as mycelium within the seed or hull. Some smuts show features of more than one type, as flag smut of wheat, which is of the seedling infection group, but in which the spores survive between crops both on the seed and in the soil, necessitating additional control measures.

SEEDLING INFECTION SMUTS: BUNT OF WHEAT

(*Tilletia foetida* and *T. caries*)

History and Distribution. Bunt, also called covered smut or stinking smut, occurs wherever wheat is grown and is one of the major diseases

of this crop. It is particularly damaging in the Pacific Northwest where the survival of bunt spores in the soil creates a difficulty in control. Before the discovery of the infective nature of bunt by Tillet in 1755 and of the germination of bunt spores by Prevost in 1807, it was commonly believed that bunt was due to unfavorable environmental influences, and it had undoubtedly been known as a major pest of wheat since ancient times. Even today there are many farmers who are unacquainted with its contagious nature. The scientific farmer, Julius Kühn, was the first to use bunt spores in infection experiments, thus showing the parasitic nature of the fungus. The use of seed disinfection for bunt control is said to trace back to 1670 when a wheat ship was wrecked off the coast of Bristol, England, and the salt-soaked grain was salvaged for seed and found to produce a bunt-free crop. Copper sulfate soaks were used for bunt control in the United States at least as early as 1858. In recent years studies on bunt have been concerned largely with its control by seed treatments and breeding for bunt-resistant varieties, the effect of environmental factors in bunt development, and physiologic specialization of the bunt fungi.

Importance. Bunt causes important losses in several ways. The field loss of grain is directly proportional to the percentage of bunt in the field which in severe cases may reach 20 per cent or higher. The smutty grain is then considered inferior in quality and its price is lower than that for smut-free grain. Wheat is graded "light smutty" if an 8 oz. sample contains 14 or more smut balls and "smutty" if it contains 32 or more smut balls or equivalent discoloration. The dockage varies from 1 cent to 10 cents per bushel, depending on the amount of smut and the prevailing price of wheat. This dockage is due to the difficulty in making a good grade of white flour from the discolored and foul-smelling grain, and to the explosion hazard in elevators and mills handling smutty wheat. The loss from bunt in the national wheat crop has progressively decreased from 4 per cent in 1926 to about 1 per cent at the present time, owing mainly to increasing adoption of seed treatments. Fluctuations in the annual losses, which may be very considerable, are largely due to prevailing temperatures at planting time. Indirectly the supply of moisture may be the deciding factor, not through any direct effect on infection, but because the availability of moisture is paramount in determining the planting date each season. In winter wheat areas, a dry fall will greatly increase the acreage of late-planted wheat, in which bunt would be favored by the cool temperatures then prevailing. There appears to be no indication that smutty wheat is either unpalatable or poisonous to livestock, and this also applies to corn and other grains which may be smutted.

Host Plants. The bunt fungi can attack rye and certain grasses, but

the disease is of practical importance only on wheat. Among the wheats all conditions are found from complete susceptibility to complete freedom from smut. All of the leading species of wheat are attacked to some degree. Varietal resistance in wheat is most important in the Pacific Northwest where seed treatment is ineffective in control because of soil- and air-borne infestation. Here the bunt-resistant varieties Relief, Wasatch, and Cache

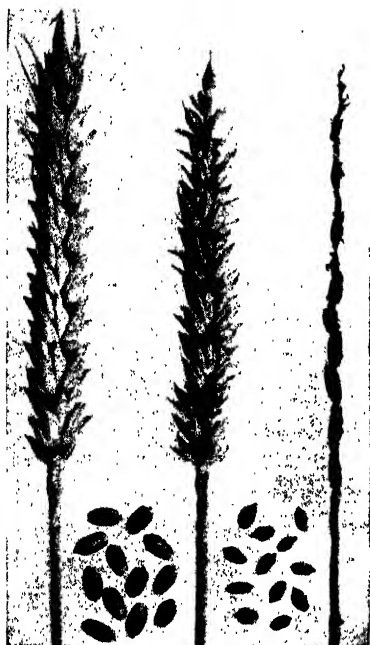


FIG. 18. The head smuts of wheat. (Left) Healthy head and grain. (Center) Bunted head and bunt balls. (Right) Loose smut head with the smut spores mostly blown away. (Courtesy, Benjamin Koehler, Ill. Agr. Exp. Sta.)

are outstanding results of recent breeding efforts. In other wheat areas, varietal resistance is less important since simple seed treatments give complete bunt control. Some strains of the Turkey variety are highly bunt resistant as are the winter wheat varieties Comanche, Pawnee, Nebred, Sibley 81, and Blackhawk, but most of the other commercial winter wheats are quite susceptible.

Symptoms and Signs. Both bunt fungi cause stunting of the wheat plant. In the case of *T. foetida*, which is the predominant bunt fungus over most of the United States, the culms are slightly shorter than healthy culms ("high bunt"), while in the "dwarf bunt" (*T. caries*), which is the leading form of bunt in the Pacific Northwest but which occurs also in other parts of the United States, the affected culms may be a foot shorter than healthy ones. Bunt-infected seedlings sometimes show a chlorotic mottling of the leaves. No other conspicuous indication of the disease is seen until

just before harvest, when the grains are found to be transformed into balls of black chlamydospores, at first greasy, later powdery (Fig. 18). Affected heads give off a foul odor, likened to that of decaying fish. Often affected heads can be quickly recognized in the field by the presence of small, shiny, black beetles which feed on the fungus material. The awns of affected heads often stand out from the head at irregular angles, and the glumes also have a tendency to stand out, giving the head a loose appearance. Affected heads are a more bluish green than

normal ones. Threshed grain containing an appreciable amount of bunt has the characteristic bunt odor, and the grains are darkened by presence of smut spores, especially in the crease and brush. Mixed with the grain will be found the short, thick bunt balls filled with chlamydospores.

Etiology. The two fungi causing bunt, *Tilletia foetida* and *T. caries*, are similar in most respects, with no essential differences in life history and control. When a smutty crop is harvested or stored, many of the bunt balls break, and the chlamydospores come to rest on the healthy grains. Here they lie dormant until the seed is planted. When the seed germinates, if temperatures are suitable, the smut spores also germinate, each chlamydospore producing a short, thick promycelium on the tip of which are borne eight threadlike sporidia which unite in pairs, producing four H-shaped sporidia (basidiospores) (Fig. 17). These in turn germinate by producing an infection thread that enters the wheat coleoptile, or by producing short, curved secondary sporidia that in turn germinate by means of an infection thread which enters the wheat seedling. As the seedling develops, the smut mycelium ramifies through the tissues, enters the blossom primordium, and replaces the ovary with mycelium which segments, each cell becoming transformed into a chlamydospore.

The bunt fungi exhibit physiologic specialization. There are 36 or more physiologic races, each capable of attacking a distinct group of wheat varieties.

Epiphytology. Bunt spores require cool soil for germination and infection, 41° to 65°F. with optimum infection at 45° to 55°F. Little or no infection occurs in warm soils at 70°F. or higher. Thus, smutty wheat which germinates in warm soil may escape the disease. Moderate soil moisture is favorable to bunt infection, from 15 to 60 per cent of the soil capacity. When wheat grows rapidly under exceptionally favorable conditions, the bunt fungus inside is sometimes unable to grow rapidly enough to invade the head completely, resulting in a partially smutted head or even in complete escape from the disease. Reports disagree as to the relation of soil type to bunt, but there is some indication that the disease is favored by sandy soils and those well supplied with humus, K, and P.

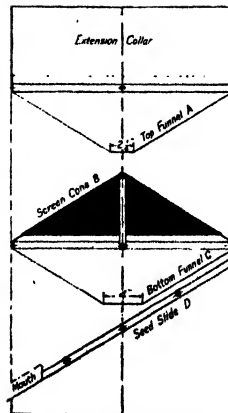
The heaviest bunt losses are associated with deep, late planting of fall wheat or early planting of spring wheat, abundant inoculum on the seed, moderate soil moisture, average growing conditions, and lack of seed treatment, or persistence of the spores in the soil, as in the Pacific Northwest.

Control. It has long been known that *seed treatments* are effective in controlling bunt. Since the disease is carried from one crop to the next by the spores on the surface of the seed, any chemical which would destroy the spores without injuring the seed might be expected to control the

disease. Formerly it was the practice to dip wheat seed in a solution of formaldehyde or copper sulfate in order to destroy the spores. More recently it has been discovered that the same result may be obtained more cheaply and easily by dusting the seed with disinfestant dusts, and today the dust method is almost exclusively practiced for the control of bunt. Various chemicals may be used for dusting seed. Among the cheapest and best, however, are copper carbonate, ethyl mercury phosphate (New Improved Ceresan), Arasan, and Spergon. The general prevalence of bunt, together with the added advantages of dusting wheat seed in improving the stand, indicate that wheat seed dusting is worth while and profitable as a routine practice. To be sure, untreated seed will sometimes escape the disease, but a cost of 2 or 3 cents per acre will provide disease



55 GAL. DRUM



CROSS SECTION

B

(See facing page for legend.)

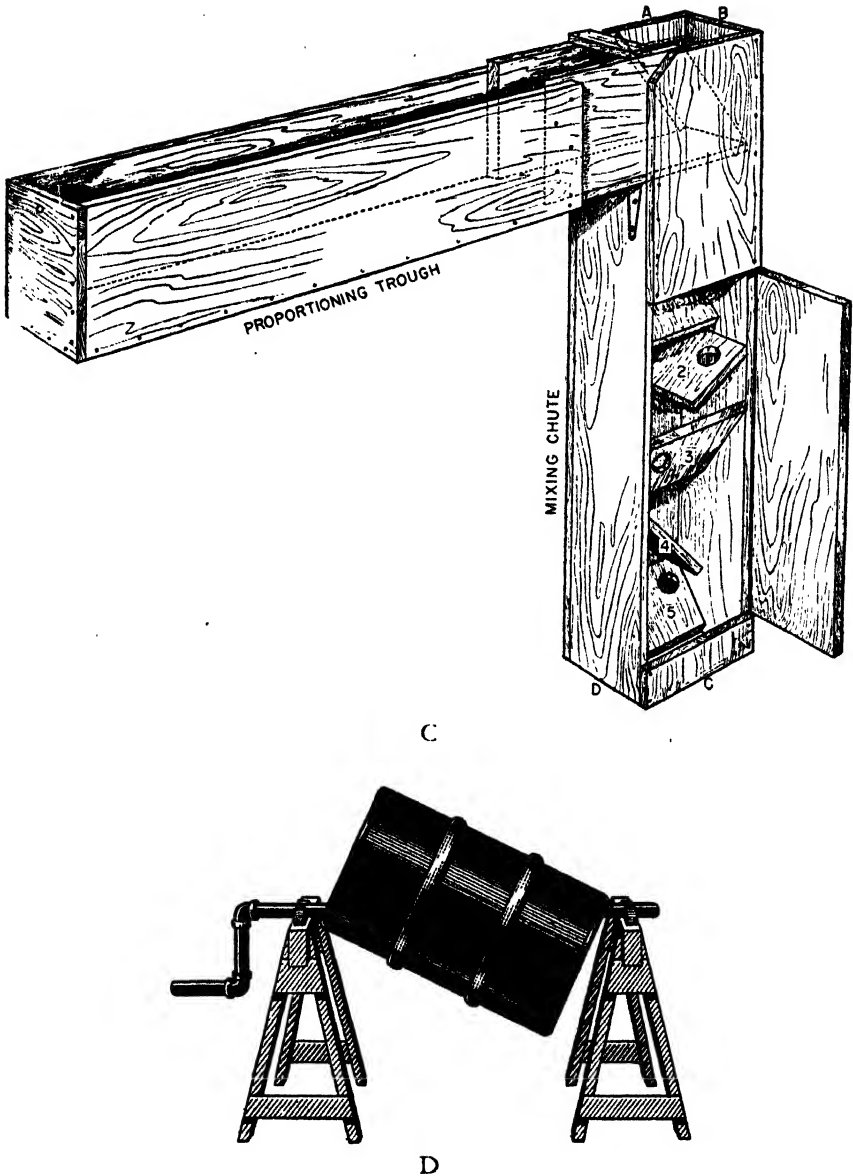


FIG. 19. (A-B) Types of treaters used in dusting field crop seed. (A) Commercial cleaner-grader-duster suitable for treating 300 to 500 bushels of grain seed per day. (B-D) Home-made treaters. (B-C) Gravity types. (D) Revolving barrel type. The oil barrel treater (B) is convenient for treating hull-less small grain seed with volatile dusts, while the Minnesota seed treater (C) is suitable for all cereal seed and for applying any standard type of dust. D, which originated at the Pennsylvania State College, is the only one of these types that can be used for treating fuzzy cotton seed.

insurance against those years in which bunt would take a heavy toll of the wheat crop. The dusting is best accomplished by the following procedure:

Before using seed treatment it is necessary to clean and grade the seed by using a fanning mill and screens in case any bunt balls are present. Seed treatments will give only partial control if bunt balls are mixed with the grain. The cost of cleaning equipment is not excessive, and the cleaning and grading will remove many smut balls, weed seeds, and shriveled kernels. Cleaned and graded seed produces cleaner, stronger plants.

For the dust treatment use either 50 per cent grade copper carbonate dust (2 oz. per bushel of seed) or one of the other recommended chemicals at the dosage rate indicated on the container, usually $\frac{1}{2}$ or 1 oz. per bushel of seed treated, making the cost for chemical in the neighborhood of 1 to 2 cents per bushel of grain. The dusting may be quickly and continuously carried out by using a gravity mixer of either of the types shown in Fig. 19.

Either of these treaters can be made at home for about \$3 and either is a great improvement over the older revolving barrel type. In using either type, pour in a bushel of seed, add the necessary amount of dust, stir in the dust for a moment, and then allow the seed to run through into the receiving sack by raising the hopper (Fig. 19C) or pulling the plug (Fig. 19B). With "Ceresan" dust either type of treater may be used, but with the other chemicals the wooden type (Fig. 19C) is preferred. The treatment does not usually cause treated grain to deteriorate in storage between crops. It may be treated at any time between harvest and planting time, and often maintains good viability if held over in storage till the second year.

Several types of power-driven seed treaters are available. Among them are treaters manufactured by:

Calkins Mfg. Co., Hutchinson, Kan. (Combination cleaner-grader-treater).

Ben Gustafson Seed Grain Machinery Co., Fargo, N. Dakota.

Clow-Winter Mfg. Co., 1117 Metropolitan Life Bldg., Minneapolis, Minn.

The United States Department of Agriculture has issued a circular (No. 415) describing the commercial treaters. Wheat seed treatment on a community basis has proven successful in a number of states, the machine being owned by a farm group or owned and operated on a custom basis by a single farmer, 4-H member, or Future Farmer. Many grain elevators and

feed and seed concerns have installed power-driven seed treaters and treat seed grain as a service to local farmers.

Note the following *precautions* in dusting seed:

- a. The dusts are poisonous. The operator should tie a cloth across mouth and nose, wear gloves, and work out of doors.
- b. Dusted grain is poisonous and should be stored where livestock will not feed on it.
- c. Some seed treatment chemicals, particularly mercury dusts, are injurious to seed if excessive amounts of dust are used. Follow directions and do not store treated seed in airtight containers.
- d. Copper carbonate has a tendency to clog the drill. After using it the drill should be carefully cleaned out. This trouble does not apply to other standard seed treatment dusts.

Wheats differ in susceptibility to bunt, and in the Pacific Northwest where soil-borne bunt is common and seed treatment ineffective, the use of *resistant varieties* is the leading control measure. Here the varieties Relief, Cache, and Wasatch have come into general use for bunt control. Certain other commercial wheats are bunt resistant (see p. 52), but as most of the important commercial varieties are susceptible and chemical control is simple and inexpensive, the use of resistant varieties in bunt control is limited largely to areas of soil-borne infestation.

OTHER SEEDLING INFECTION SMUTS

Insofar as life history and control are concerned there are few differences between bunt and the other seedling infection smuts listed on p. 50, but certain of these differences are worthy of mention.

Oats and Barley. The seeds of oats and barley are protected by a hull. The seedling infection smut fungi attacking these crops (Fig. 20) may penetrate the hull and live between crops not as spores on the seed surfaces but as mycelium within the hull. On germination of the seed, this mycelium starts into activity and grows into the seedling. A chemical that is not volatile, such as copper carbonate, Spergon, or Arasan, fails to penetrate the space between hull and seed, and hence does not protect the seed. For this reason volatile fungicides, such as formaldehyde or organic mercury dusts are required with these smuts. Formaldehyde treatments have long been used (Fig. 21), but more recently Ceresan is replacing formaldehyde to a considerable degree. The dust is applied as in the case of bunt, except that the wooden, gravity type of treater or



FIG. 20. Loose smut of oats. The spikelets may be almost entirely converted into black spore masses or, in many cases, as at the right, the glumes are only partially destroyed. (Courtesy, Illinois Natural History Survey.)

the rotating barrel type is preferred to the oil-barrel gravity treater which does not allow a free flow of seed. If formaldehyde is used it may be applied in any one of three ways. In all three cases the same amount of formaldehyde is used, namely, 1 pt. to 50 bu. of seed.

Concentrated formaldehyde mixed with an equal amount of water may be sprayed on the grain as it leaves the grain spout or is shoveled from one heap to another, after which the grain is covered with a tarpaulin for four to eight hours and then aerated. Less convenient are older methods by which the grain is soaked with dilute formaldehyde and then sown immediately or dried before storing.

There are at least 44 races of the two similar oats smut fungi, *Ustilago avenae* and *U. levis*, but plant breeders have been successful in combining resistance to many of these races in agronomically desirable oat varieties, such as Clinton, Cedar, Mission, Florilee, Ventura, Traveler, Osage, Markton, Uton, and Navarro, most of which are also rust-resistant.

Sorghum. Sorghum suffers from two common smut fungi, covered smut (*Sphacelotheca sorghi*) and loose smut (*S. cruenta*), with life histories and control similar to that of bunt (Fig. 22). There is a third disease,

sorghum head smut, but this is too rare to warrant special efforts at control. Sorghum, being a feed crop, frequently receives less care from growers than cash crops, and sorghum smut often has been allowed to destroy much of the crop. This is unnecessary, as sorghum smut can be very easily and inexpensively controlled by seed treatment.

Sorghum seed is somewhat more sensitive to chemical injury than other seed but there is no danger in the use of standard seed disinfestants if overdosage is avoided. In the past, copper carbonate dust has been used extensively but more recently there has been a tendency to shift to the newer organic dusts, such as Ceresan, Arasan, and Spergon, which can be applied with a gravity-type, rotary, or power-driven seed treater, as in the case of bunt. A volatile dust such as Ceresan must be used on those types of sorghums with persistent glumes.

Flag Smut of Wheat (*Urocystis agropyri*). Primarily a leaf and stem disease, flag smut of wheat persists between crops both on the seed and in the soil. For this reason seed treatments are not sufficient for control, and this is accomplished by the combination of seed treatments, the use of flag smut-resistant varieties (compulsory in the area of infestation in the central states), and crop rotation. Flag smut of wheat is a very destructive disease but fortunately is limited in the United States to a small area in the

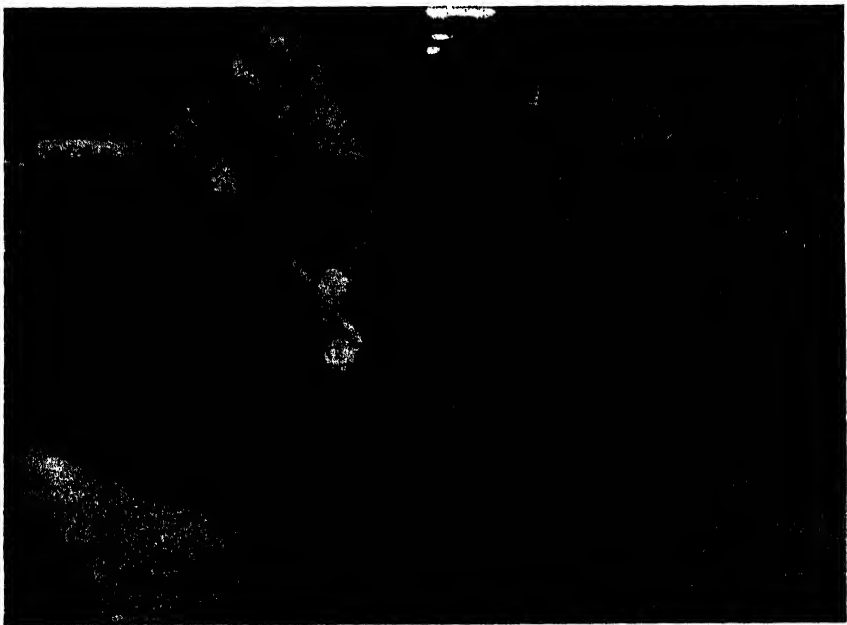


FIG. 21. Spraying oats with formaldehyde for controlling loose and covered smuts. (Courtesy, R. S. Kirby, Pa. Agr. Extension Serv.)

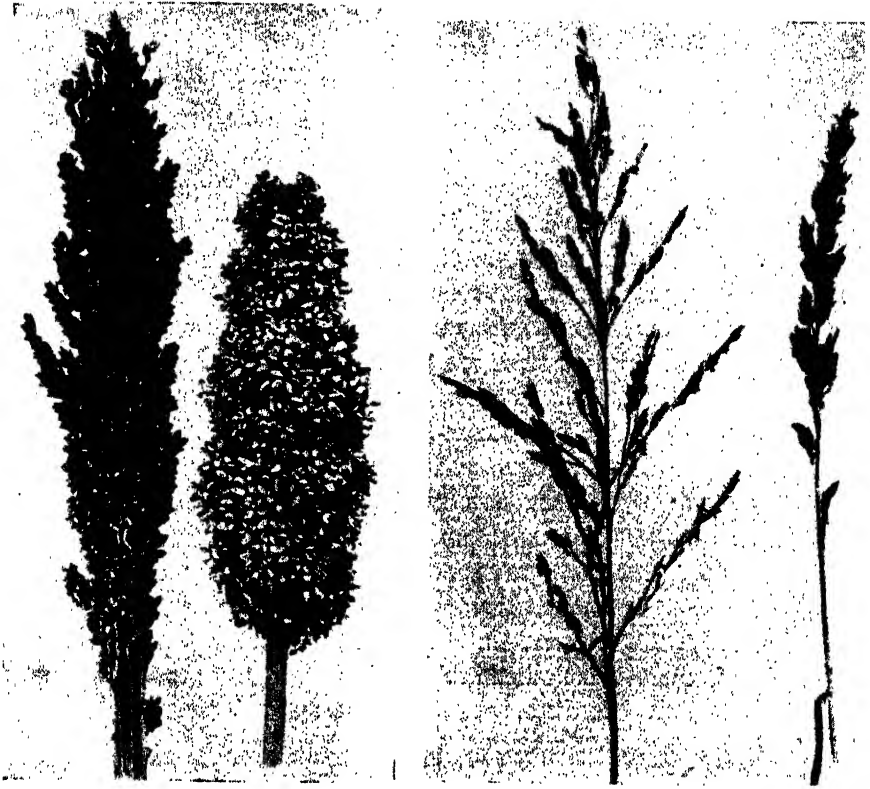


FIG. 22. Sorghum smuts. (Left to right) Normal head of grain sorghum; grain sorghum with covered kernel smut, the grains being replaced by white horns filled with black smut spores; normal Johnson grass (a wild sorghum); Johnson grass with loose kernel smut.

central states and the Pacific Northwest. It was first found in Mexico in 1945. In addition to wheat, several species of grasses are affected. There is a law forbidding importation of unprocessed wheat from Australia, Asia, South Africa, Italy, and Spain, designed to protect American growers from further introductions of flag smut.

Rye. Rye is affected, though rarely, by both the wheat bunt and loose smut organisms. The more common and serious smut of rye is a stem and leaf disease caused by *Urocystis occulta*, kin to the flag smut and onion smut fungi. The disease is recognized as black, elongated sori on stems, leaves, and heads, ending in destruction of the entire plant with complete loss of bearing. For the most part the inoculum survives on the seed where it is amenable to chemical disinfestation, although in some localities and dry seasons soil-survival may occur, calling for crop rotation as a supplementary control measure.

BLOSSOM-INFECTION SMUTS: LOOSE SMUT OF WHEAT
(*Ustilago tritici*)

History and Distribution. Loose smut or "blackheads" of wheat, is worldwide in its distribution, being most serious in regions with humid weather during the blossoming period, regardless of the total annual rainfall. The disease has been known since early times, but only during the past 50 years has the distinction between loose smut of wheat and the smuts of oats and barley been recognized; also, the life history and control of loose smut have been worked out only recently, beginning with the separation of the barley smuts by Jensen in Copenhagen (1888). The important discovery of blossom and internal seed infection was made by Maddox in Tasmania (1895-1897) and at once confirmed by several Japanese workers. Jensen, recognizing with others that chemical seed treatments were ineffective for loose smut control in wheat and barley, first suggested a hot water treatment for this purpose (1887-1889). Recent work has concerned itself mainly with varietal resistance toward loose smut, genetics and specialization of the fungi involved, and improvements in the hot water treatment.

Importance. While loose smut is a major disease of wheat, in past years the estimates of its damage have ranged somewhat lower than those for bunt, the national loose smut losses usually being between 0.5 and 1 per cent of the crop. Bunt has been decreasing progressively in importance during the past decade, thanks largely to widespread adoption of seed dusting, while loose smut has constantly increased until its importance is equalling or even exceeding that of bunt in many areas. In Nebraska in 1945, for example, an extensive survey revealed loose smut as the most destructive wheat disease that year, causing an average loss of 6.5 per cent of the crop. While average loose smut losses in the main Wheat Belt range from 1 to 4 per cent, individual fields with 5 per cent or more are not uncommon, and fields with as much as 40 per cent infestation have been recently observed in this area. As in the case of bunt, affected plants usually are a total loss, and the percentage loss in yield closely agrees with the percentage of infestation in the field. Also, loose smut infection has been shown to increase winter killing. The grower is inclined to underestimate the loose smut loss or attach little importance to it because the disease is inconspicuous at harvest time and the harvested grain is uninjured in quality. For the latter reason the elevator operator, who is largely responsible for the bunt control program, enforced by dockage penalties, puts no pressure on the grower with regard to loose smut.

Host Plants. *Ustilago tritici* is important only on wheat, although rye

and barley can be attacked by this fungus. Various degrees of resistance and susceptibility occur in different wheat varieties. While many of the commercial varieties are highly susceptible, some degree of resistance has been seen in the varieties Kawvale, Early Blackhull, Bacska, Forward, Fulcaster, Leap, Redit, Trumbull, Fulhio, Illinois No. 2, Red Chief, Redhart, Valprize, Cooperatorka, Kanhull, Nabob, Pawnee, Hussar, and Hope-Hussar hybrids. Some of these are field-resistant but show susceptibility to one or more loose smut races.

Symptoms and Signs. The disease is most apparent in the field at the blossoming period, when affected heads become transformed into dark, powdery masses of fungus spores (Fig. 18). All parts of the head are destroyed, and the spores soon shatter away, leaving only the naked rachis, which is inconspicuous at harvest time. Affected stems usually are shorter than normal ones. Generally, but not always all stems from a single stool are affected. The harvested grain from fields containing loose smut is not discolored or lowered in quality; the loss is entirely one of yield and value for planting purposes.

Etiology. The chlamydospores of *Ustilago tritici*, the fungus that causes loose smut, mature on the affected heads as adjacent normal heads are blossoming. They are carried by wind to the healthy heads, where they lodge on the feathery stigmas and germinate much as a pollen grain. The infection thread grows down through the style into the ovule, and as the ovule develops into a seed the fungus mycelium occupies the embryo, where it lies dormant until the seed germinates. Infected seed are indistinguishable in appearance from normal seed but can be recognized by microchemical tests. As the seed germinates, the fungus resumes activity, grows up inside the developing head, reaches and invades the flower primordia, and replaces the floral parts with masses of chlamydospores. By a nice adaptation, the infected heads emerge just before normal heads, so that dusty masses of spores are available to infect the normal heads as they emerge.

Thus the smutty heads observed this year came from normal looking seeds that were infected at blossoming time last year but gave no evidence of their infection until a full year after infection occurred. Spores in the soil or on the surface of seed are of no consequence in the life history of this smut; they are delicate and soon die.

Ustilago tritici consists of seven or more physiologic races, each of which is limited in its attack to certain varieties of wheat.

Epiphytology. Loose smut is most prevalent during years when moisture and inoculum were adequate at blossoming time the previous

year. From this, its severity can be predicted with reasonable accuracy a year in advance. The structure of the blossom appears to have little correlation with susceptibility, although in barley loose smut escape is correlated with the closed type of flower. Time of planting appears to have an effect on the amount of infection in winter wheat, late planted wheat showing the least infection, while the greatest infection is seen in wheat planted at normal planting dates. Heald explains this on the assumption that in late plantings at low temperatures, the fungus fails to come out of its dormant state when the seed germinates, and the plant thus "runs away from the fungus." Rapidly growing wheat may escape injury, even though infected, if the fungus inside is unable to develop fast enough to reach the floral parts.

Control. Since the loose smut fungus is *inside* the seed, dusting or any other method of sterilizing seed on the surface will not control it. The only effective method of disinfecting wheat seed which contains the loose smut fungus is to heat the seed to a temperature that will destroy the fungus inside the seed without injuring the seed itself. Since the treatment requires considerable care, it is suggested that growers plan to treat only enough seed for a seed-production block (5 to 10 per cent of the main wheat acreage), and harvest the grain from this treated block for seeding the main acreage the following year. The most effective plan is for growers to cooperate in the treatment, rather than to carry out the treatment independently on individual farms.

Where special equipment for hot water seed treatment is not available, the following method can be used.

Before treating, the grain should be well cleaned and graded. Put the seed into coarse burlap sacks, filling the sacks about half full. Tie the sack openings so as to give the seed ample room to swell. It is good practice to put a measured bushel of dry seed into each sack, if the seed is to be sown wet. The seed will swell considerably and this will make it easier to sow at the desired rate. Provide water in containers such as water tanks or other large vessels. A pond with hard bottom and clean water or a river may be used, provided the water is deep enough to cover the sacks well. Lay the sacks in the water on their sides, not on end, and leave them to soak four or five hours. Caking of the grain when it begins to swell should be prevented by moving the sacks two or two and one-half hours after they were placed in the water; this stirs the grain.

- The following equipment is required: three barrels or other containers such as tanks or tubs, two containers for the heating of water, a heater or a supply of live steam from an engine or boiler, and a reliable thermometer,

one that will float being most convenient. Also, it is well to have some sort of a pulley arrangement over the barrels or containers for lifting the sacks from one bath to another. The arrangement is shown in Fig. 23.

Fill the first barrel about two-thirds full of warm water at 120°F . Fill the second barrel two-thirds full of hot water at 129° . Then fill the third barrel almost full of cold water. If live steam is not available keep a reserve supply of about 10 gal. of boiling hot water, from which the first two barrels may be replenished from time to time as they cool below the required temperatures. It is well also to have at hand a supply of cold water for the purpose of cooling the water in these barrels, if this should be necessary. If a rope and pulley are used, tie the rope to the top of one of the sacks of grain which has been soaked and drained, and tie another short rope, 3 or 4 ft. long, to one of the lower corners of the sack. Plunge the sack into the first barrel containing the warm water and leave it in about a minute. This will warm the seed and prevent too much cooling of the water in the next barrel. Move the sack about while in the water in order that the seed at the middle of the bulk will be reached. This can best be done by lifting with first one rope and then with the other. The water in the second barrel will be too hot for the hands and stirring of the seed will be impossible unless the ropes are used. Lift the seed from the first barrel or warming bath, drain for a few seconds and plunge it into the second barrel which contains the hot water. Here it should be moved

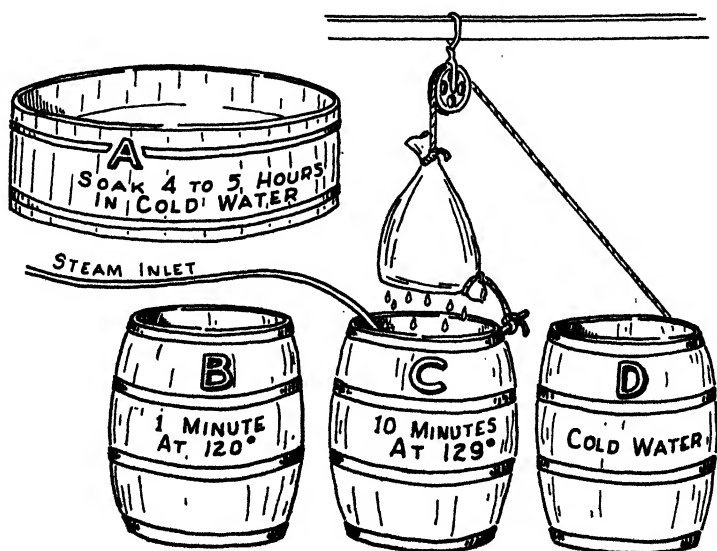


FIG. 23. A simple arrangement for the hot water treatment of seed wheat for loose smut control. (After Brentzel.)

about, as in the first barrel, and left exactly 10 minutes. The temperature throughout this 10-minute period must be held as near the 129° mark as possible. As the water cools it will be necessary to add hot water or steam and to stir well. If the temperature should drop to 124° the addition of 1 gal. of boiling water would raise the temperature almost 2°. At this ratio it would be necessary to add about 2½ gal. of boiling water to bring the temperature up to the 129° mark. When the seed has been in the hot water exactly 10 minutes draw it out and plunge it immediately into the third barrel which contains the cold water. This will stop the action of the heat and thereby avoid injury to the seed which would be sure to occur if the process were prolonged. If the grain can be spread out in a thin layer immediately upon coming from the hot water it will not be necessary to cool it by dipping it into the cold water.

As in the case of dusting, the hot water treatment for wheat seed has worked out very well on a community basis, which minimizes the investment in equipment and keeps the cost of treatment down to a small amount. The principal features of the equipment are a boiler and tanks, and various communities have worked out ingenious methods of obtaining this equipment, such as the use of a steam tractor, or adapting a creamery, cotton gin, mill, or laundry to the seed-treatment work. Since hot-water-treated seed does not deteriorate in storage the treatment can be carried out at any time in the off-season.

To simplify the hot water treatment a hot water seed treatment machine has been developed at the Oklahoma Agricultural Experiment Station and put into practical operation (Fig. 24). The machine has a capacity of treating 500 bushels of seed grain per day, and combines the advantages of automatic time control, semi-automatic temperature control, efficient penetration of heat, and portability. It is transported from one community to another, serviced with steam, electricity, and water from cotton gins, elevators, creameries and the like, and operated on a self-sustaining custom basis at a charge of 5 to 10 cents per bushel of grain treated. The grain is presoaked by the farmers and after treatment is dried on roped-off pavement or on the farm.

Because the hot water treatment is laborious and requires special equipment, many growers faced with a loose smut problem may find a more satisfactory solution in discarding the smutted crop for seed purposes, and starting fresh with certified seed or seed known from inspection of the seed crop at blossoming time to be free of loose smut.

Control of loose smut is effected also by the use of the more resistant wheat varieties, several of which are listed on p. 62. It must be borne in mind that with any organism which exhibits physiologic specialization,



FIG. 24. Oklahoma hot water equipment for treating wheat and barley seed grain for loose smut control. Preheating tank in foreground, main tank at the rear. Each basket holds a bushel of grain. (Right) An electric motor activates endless belts at the bottom of the tank, which move the baskets the length of the tank in 10 minutes (wheat) or 13 minutes (barley), giving automatic time control. The water is heated by free steam manually controlled by the ball valve at the left.

resistant varieties may from time to time be attacked by new physiologic races of the pathogen. This limits their period of usefulness and necessitates constant efforts at their replacement with newer resistant varieties.

BLOSSOM-INFECTION SMUTS: BROWN LOOSE SMUT OF BARLEY (*Ustilago nuda*)

Barley is subject to three smut diseases: covered smut (*Ustilago borderi*), black loose smut (*U. nigra*), and brown loose smut (*U. nuda*). The first two are similar to bunt and are controlled by seed dusting, using a volatile dust that will penetrate the hull-bearing seed. Brown loose smut has a life cycle similar to that of loose smut of wheat and requires a hot water treatment for control. As barley seed is somewhat more susceptible to heat injury than wheat seed, barley is treated at 126°F. for 13 minutes instead of at 129° for 10 minutes as in wheat. It is not easy to distinguish the two loose smuts of barley in the field, and this is further complicated by the fact that loose smut is sometimes caused by a mixture of *U. nigra* and *U. nuda* or, more rarely, by hybrid smuts. A simple laboratory spore germination test serves to distinguish these smuts. If the spores all germinate to form masses of secondary spores (sporidia), the smut is *U. nigra* and can be controlled by seed dusting. If some or all of the spores germinate to form mycelium, the blossom-infection type of life history is indicated,

and hot water treatment will be needed. A recent survey has shown that about one-half of the barley loose smut in America is of seedling infection type, subject to control by seed dusting.

LOCAL INFECTION SMUTS: COMMON SMUT OF CORN

(*Ustilago zeae*)

History and Distribution. There are several smut diseases of corn of which common or boil smut is by far the most important. The head smut of sorghums also attacks corn but is much rarer than common smut. The common smut is world wide in its distribution wherever corn is grown except in Australia where it has been eradicated. The disease has been known since very early times and has probably existed in America for centuries. At first it was thought that smut was an exudation of superfluous sap from highly nourished plants. In the last half of the past century its parasitic nature was realized but it was thought to be seed-borne. Not until 1895 was it finally recognized that corn smut differs from the other smuts in that it is not seed-borne and that it is distinguished by the fact that infections are local and not systemic. Recent work on corn smut in the main has concerned attempts at breeding smut-resistant varieties of corn.

Importance. Smut is a major problem wherever corn is grown in America. Average annual losses for the United States are usually from 3 to 5 per cent, or 50 to 80 million bushels, the losses in individual states ranging up to 10 per cent and in individual fields up to nearly 100 per cent. Both field and sweet corn are severely attacked. The losses from smut are difficult to estimate because of the nature of the disease, attacking as it does any aboveground portion of the plant. The damage includes yield reduction through sterility, destruction of the ears, lodging, reduction of the amount of fodder, and lowering of the carbohydrate content. Smutted stalks yield about one-third less grain than healthy stalks. In sweet corn the loss is increased by the fact that an ear which is smutted to only a minor extent is still a total loss for use as a roasting ear.

Host Plants. The corn smut fungus attacks only corn and the closely related teosinte. There is considerable variation in susceptibility among the different varieties of corn, but in controlling smut relatively little use has been made of this fact because of the difficulties arising through physiologic specialization of the smut fungus, and because in many cases resistance has been found to be correlated with lack of vigor in the corn plant which is even less desirable than smut susceptibility. Sweet corn is most susceptible, field corn next, and popcorn least of all. Of the field corns, the flint varieties are more susceptible than the dent

varieties, and some of the commercial hybrids of the Corn Belt are relatively resistant.

Symptoms and Signs. The fungus causing common smut can attack any aboveground organ of the corn plant: stalk, prop roots, leaf, tassel, husk, or ear. The disease is readily identified by the boils or masses of black chlamydospores which are often very large on ears or stalks and much smaller on leaves or tassels (Fig. 25). At first the boils are covered



FIG. 25. Corn smut. Boils like these appear on any aboveground part of the plant, often lowering the yield of affected plants by 30 per cent or more.

with a greenish-white, firm, glistening membrane. In this stage they are edible, and are sometimes served, boiled or fried, as a rustic delicacy. As the boil matures, the membrane dries and weathers away, releasing myriads of dark spores. In ears or tassels, only a part of the organ is involved in the boil, in contrast with head smut. The plant often is remarkably distorted or abnormal. A boil at a node may result in a stem that bends at a right angle and becomes horizontal. Infected tassels often develop normal kernels, the central spike of the tassel resembling a small

ear. In the related disease, head smut, leafy shoots develop in the tassel, and the smut masses in the ear contain stringy fibers which become conspicuous as the spores are blown away. These two features are not seen in the case of common smut, and their presence or absence distinguishes the two diseases.

Etiology. Common smut is caused by the fungus *Ustilago zaeae*. Distinct from most of the smut fungi, *U. zaeae* causes only local infections; it does not pervade the entire plant, and each boil results from a separate, distinct infection. The fungus is not seed-borne and does not infect the germinating seedling. The chlamydospores overwinter in the soil, in corn debris, or in manure or litter from livestock that have fed on smutty corn. Under favorable conditions the chlamydospores germinate and produce sporidia, which in turn can multiply by budding to produce secondary sporidia (Fig. 26). When the corn plant is 1 to 3 ft. high, some of these sporidia are carried by the wind and other agencies to the younger tissues of the corn plant, especially the moist funnels at the leaf bases, and here germinate to produce infection hyphae which penetrate the corn tissues. Mycelium from spores of two sexes must be present if the fungus is to develop actively. As the infection progresses, the boil appears, at first permeated with mycelium, later filled with chlamydospores formed by the rounding off and separation of individual cells of the hyphae. The membrane dries and ruptures, and the spores are liberated. They may be blown about or may germinate at once, in the boil, producing sporidia which initiate new infections, especially in the ears. Eventually, the chlamydospores are returned to the soil where they lie dormant during the winter. The smut fungus exists as many physiologic races, each capable of attacking only certain varieties of corn. Sexual reproduction occurs, which enables these races to hybridize with one another, and mutant races are constantly appearing, giving a great variety of distinct smut lines or races.

Epiphytology. Corn smut is adapted to warm weather. The optimum temperature for spore germination is 80° to 92°F. which is considerably higher than for many other smuts. Heaviest infection occurs when scant rainfall in the early stages of corn development is followed by moderate rainfall as the crop approaches maturity; the least infection is associated with heavy rain in the early part of the season, followed by dry weather. Light but evenly distributed rains throughout the growing season usually result in moderate infection.

Spore germination in the soil is favored by an acid soil reaction, the optimum being pH 4.9. Susceptibility to smut is increased in weak plants of low vigor by stimulating their growth with fertilization or cultivation, but the most vigorous, rapidly growing plants, while highly susceptible to

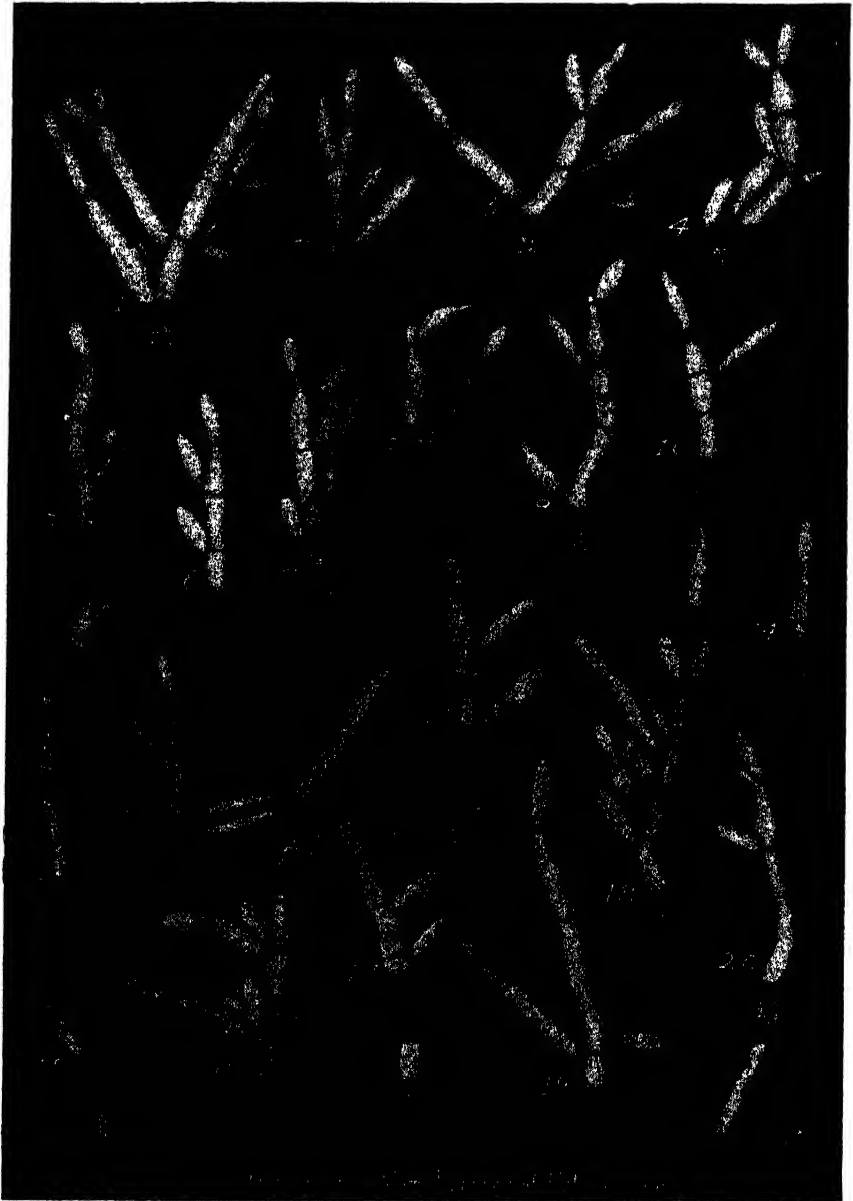


FIG. 26. Germinating chlamydospores of corn smut, illustrating deviations from the normal four-celled promycelium. (Courtesy, M. F. Kernkamp and M. A. Petty, Minn. Agr. Exp. Sta.)

smut, may largely escape its most serious effects through their rapid growth. Corn varieties with tight, long husks are less affected by smut than varieties with loose, open husks.

The thick-walled chlamydospores are quite resistant to aging, and will retain their viability for as much as five to seven years. They are destroyed by digestive juices of the alimentary tract or by the acids which accumulate in silage. The sporidia, on the other hand, are thin-walled and less resistant than the chlamydospores.

Smut is reported to be less serious in well spaced hills with two or three plants per hill than in closely spaced hills, or hills with more or fewer stalks per hill.

On the whole, corn smut is a type of disease which appears fairly regularly to about the same extent, year after year, and is not subject to marked fluctuations in importance from one year to another.

Control. No seed treatment is effective against corn smut. The general use of dust treatments for corn seed has to do with seedling disease and stalk, root, and ear rots, rather than with smut. Despite extensive corn breeding programs in many states, there has been relatively little progress in developing smut-resistant corn varieties. This is not due to lack of effort, but to the great variability in the corn smut fungus, and to a tendency for smut resistance to be associated with weak types of corn. Several investigators have found smut resistance consistently present in certain inbred lines of corn, but at present smut-resistant varieties are not available for general planting, although progress is being made in incorporating smut resistance in the newer hybrids.

For controlling this disease, the grower must turn to cultural methods—rotation and sanitation. These will not give complete control, but will materially aid in reducing losses from corn smut. A three-year rotation is recommended normally, using any crop other than corn two years out of three. Stalks of smutty corn should be either harvested or plowed under in the fall. Manure from animals that have fed on smutty corn will contain an abundance of smut spores, and this should not be returned to corn fields. Ensilage is a good means of disposing of smutty corn stalks as the spores are killed in the silo within a few weeks. Collecting and burning the smut boils as soon as they appear is helpful in small sweet corn plantings, but is usually regarded as impractical on large field corn acreages, though this point is open to question. In New Jersey, where sweet corn smut research has been particularly active, new resistant sweet corn varieties are being perfected, and it has been found that ear smut can be significantly reduced by use of insecticides such as rotenone, nicotine, and DDT.

LOCAL INFECTION SMUTS: ONION SMUT (*Urocystis cepulae*)

While this is a disease of importance only in onions grown from seed in cool climates, it is introduced because of its unusual method of control. The disease causes a smutty disintegration of bulbs and leaves (Fig. 27).



FIG. 27. Onion smut.
(Courtesy, J. C. Walker,
Wis. Agr. Exp. Sta.)

The causal organism, *Urocystis cepulae*, is capable of living in the soil for many years, but can attack only seedlings, not the plants developing from onion sets. In the past effective control has been obtained by treating the soil with formaldehyde at planting time. The planter is equipped with a reservoir for the formaldehyde, which drips into the furrow where it disinfests a small mass of soil about the seed and thus protects the seedling during its susceptible early growth period. More recently this has been largely replaced by treating the seed with very heavy doses of the newer organic seed disinfestants such as Arasan, Fermate, and Tersan; in some cases fungicides are incorporated in a plastic coating applied to seed, by means of which more fungicide will adhere to the seed than is usually the case.

LOCAL INFECTION SMUTS: WHITE SMUTS (*Entyloma species*)

White smuts, caused by species of *Entyloma*, occur on numerous kinds of plants including dahlia, water lily, spinach, poppy, buttercup, larkspur, calendula, meadow rue, beet seed capsules, sunflower, and members of the grass and nightshade families. They are rarely if ever important enough to warrant attempts at control.

The white smuts occur mainly on leaves where they cause leaf spots of various types, usually chlorotic, then necrotic. The yellowish chlamydospores are buried in the leaf tissue and germinate there, protruding their promycelia in clusters through the stomata where white masses of rodlike basidiospores are liberated; these resemble conidia of some of the imperfect fungi.

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Chapter 5

Diseases Caused by Basidiomycetes: Fleshy Fungi; Mycorrhizae

The remaining basidiomycetes, apart from the rusts and smuts, are distinguished for the most part by conspicuous fleshy or woody reproductive bodies, mushrooms, crusts, or "conks," (Fig. 28) clothed with a spore-bearing layer (*hymenium*) of club-shaped *basidia* each bearing at its tip four *basidiospores*. (Fig. 29.) Ordinarily no other spore stage is involved in the life histories of the higher basidiomycetes. The mycelium frequently shows little bridges (*clamp-connections*) connecting adjacent cells (Fig. 30).

In plant pathology the fleshy fungi or *hymenomycetes* are of particular interest in connection with wood and root decays, *Rhizoctonia* damping off of seedlings and potato black scurf, and symbiotic (mycorrhizal) relationships between tree roots and fleshy fungi without which normal tree growth is impossible.

Wood Decay

The wood rots of standing trees and fallen timber in forest and woodlot, and the decay of fence posts and construction timbers are familiar to everyone. Nearly all of this decay is due to the action of hymenomycetes, the bracket-, crust-, or mushroomlike fruiting bodies of which are commonly found on the surfaces of decaying wood. In the main these fungi are saprophytic in their habits. Saprophytic nutrition is involved even in destroying the heartwood of living trees, since the heartwood of a normal tree is composed largely of dead cells. Few of the wood decay fungi can attack and penetrate through living wood, but must enter trees through wounds or branch stubs which expose an infection court of dead heartwood. Once a tree is dead this saprophytic feeding continues until the wood is entirely disintegrated and its components are returned to the soil. The lack of aggressiveness of the wood decay fungi is more than compensated for by their enzymatic activity. Through the production of a variety of enzymes they are able to digest the chemically resistant elements of wood into simpler, chemically active and nutritive substances. The types of enzymes produced determine the type of ensuing decay, resulting

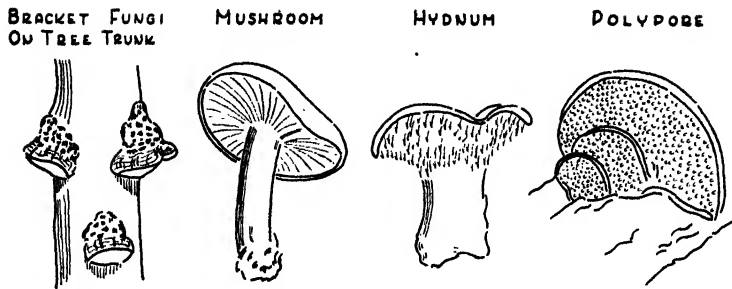


FIG. 28. Common types of fruiting bodies of the higher basidiomycetes.

in firm, white rots, dark friable rots, pocket rots, and many other types. Each fungus on each kind of wood produces a characteristic type of decay.

In addition to complete or partial rotting, the hymenomycetes and certain of the other fungi produce staining or discoloration in timber. Redstain and related troubles may not impair the strength of the wood but reduce its market grade, as stained wood cannot be used for certain pur-

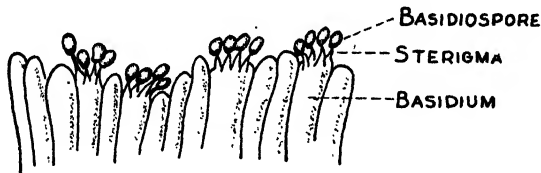


FIG. 29. Typical sexual reproduction in the higher basidiomycetes. These palisadelike layers clothe the gills, pores, or spines of fruiting bodies such as are shown in Fig. 28.

poses because of market prejudices. For many years redstain pine was not acceptable for railroad ties until the Canadian Forest Products Laboratory showed that it was as strong and durable as unstained wood.

Wood decay often is associated with injury by borers or termites, the insects and hymenomycetes coöperating in the destruction of the



FIG. 30. Various types of clamp connections seen in the higher basidiomycetes.

wood. Insects may provide points of entry for the wood decay fungi, or aid in spreading them from one tree to another.

From the fruiting bodies which are abundant on fallen decaying wood are produced countless basidiospores, providing an abundance of inoculum, particularly where such wood is allowed to accumulate and is not cleared away. J. H. White has calculated that a single conk of one of the

wood decay fungi (*Fomes applanatus*), with an area of one square foot, produces 30,000,000,000 spores in a single day and that this may continue day after day for six months or more.

Melhus and Kent cite some significant figures regarding the losses from wood decay. For example, 900,000,000 new fence posts are set each year, the majority of which are replacement of decayed posts. The Santa Fé Railroad in a single year saved 125,000,000 ties through the use of decay-control measures and this corresponds to a saving of over 2 million acres of woodland. Furthermore, we cannot disregard the prodigious losses from wood decay in standing timber. Already three-fifths of the virgin forest has been cut; we are cutting or burning 12 million acres of forest each year and only deliberately reforesting 36,000 acres. Natural reforestation helps to repair the loss, but conservation of the remaining timber is imperative and in the conservation program the reduction of wood decay constitutes a major item.

Hardly a species of tree is immune from the attack of one or more species of wood decay fungi. The fungi themselves may be restricted in host range to one or a few species of trees, or may be capable of attacking many species. The majority of the wood decay fungi can be distinguished as attacking only hardwoods (deciduous trees) or only softwoods (conifers), but there are a number that attack trees of both categories. Out of the many wood decays which occur, that caused by the fungus *Fomes igniarius* is selected for detailed treatment because of its prevalence and importance and because it has been studied more intensively than many of the other decays.

WHITE TRUNK ROT (*Fomes igniarius*)

Distribution and Importance. *Fomes igniarius* has been known for 200 years or more. It occurs throughout the entire north temperate zone, in the United States from Alaska to the Bahamas, and in South America as well. This unusual distribution is doubtless related both to its ability to tolerate a wide range of temperatures, and to its extensive host range. According to Boyce, *Fomes igniarius* causes more loss than any other wood destroyer of hardwoods. In aspen, one of the more important hosts, up to 30 per cent loss may be experienced during the first 70 years of forest growth, and even trees only 30 years old may suffer as much as 15 per cent destruction of their heartwood. It has been called the most dangerous wood decay organism of fruit trees. In some affected forests, susceptible species may be so severely attacked that hardly a single healthy, sound tree can be found.

Host Plants. The attack of *Fomes igniarius* is limited to broad-leaved

trees. Of the long list of susceptible species, among the important hosts are aspen, birch, beech, butternut, apple, pear, maple, hickory, black walnut, and alder.

Symptoms and Signs. In the earlier stages of infection no external symptoms or signs are observable because the fungus attacks the vital sapwood last of all. Ordinarily the first indications of trouble are either the breaking over of the tree, or the appearance of fruiting bodies, especially at wounds or branch stubs. These are hard, woody, hoof-shaped or less often shelflike conks from 2 to 3 in. up to 8 in. or more in width (Fig. 31). The upper surface is dull gray to black becoming cracked and rough with a smoother brownish margin. The lower, or pore-bearing surface is brown, with many tiny pore openings. The conks are perennial, i.e., they add a new layer of spore-bearing tubes each season. They may attain an age of 40 to 50 years or more. The interior of the conk is rusty brown, showing the old layers of tubes with white-stuffed pores above the present spore-bearing layer. Presence of a conk usually indicates very extensive decay within the tree.

The rot caused by *Fomes igniarius* is a firm, white decay that may extend many feet up and down the heartwood. The tree does not become hollow from this decay alone. A cross section of well-advanced decay shows in the center a white, soft, crumbly core, surrounded by one or more black lines enclosing less advanced stages of decay, which in turn are surrounded by a margin of discolored but firm wood, marking the region of advance of the fungus. Wood in the advanced stages of decay is quite useless for construction purposes, although it may still have value for manufacture of paper pulp.

Etiology. The walls of the tiny pores (0.15 mm. in diameter) are lined with countless basidia interspersed with bristles or setae, each basidium bearing four very small (6 to 7 μ) globose, colorless basidiospores. These are shot out from the sterigmata (their points of attachment to the basidium) to the center of the pore, where they drift down and on leaving the conk are picked up by the wind and widely dispersed. On coming to rest on a suitable infection court (branch stub or wound in a susceptible tree), they germinate, the infection thread enters the wood, and begins mycelial development. The mycelium advances rapidly up and down, less rapidly in a horizontal direction, digesting and feeding on the wood elements and thus inducing decay. After a prolonged period of feeding, masses of hyphae grow to the surface where there is exposed dead wood, and combine to form a solid mass of fungus tissue which develops on its under surface the first layer of pores. Each season a new pore layer is added to the underside of this conk.



FIG. 31. (A) *Fomes igniarius* fruiting bodies at old knotholes on living aspen. (Photograph, G. G. Hedgcock); (B) *Fomes igniarius* on living black ash. A single large fruit usually forms on an infected tree of most of the hardwoods except the aspens. (Photograph, H. G. Eno); (C) Rot of *Fomes igniarius* in living maple trunk. Note the broad dark zone between the healthy wood and the decay; an old knot-hole at the right is the point at which the fungus first entered, and the black mass at the right is a developing fruiting body. (Courtesy, New England Sec. Soc. Am. Foresters, Leaflet 20.)

Fomes igniarius shows a primitive type of physiologic specialization in that it contains three or more subtypes each of which shows differences from the others in appearance and behavior in culture; these are correlated with species of host trees. Cultures from single basidiospores are haploid. When two haploid cultures of different sources are grown on the same Petri dish, at the line of meeting of the mycelia there is an exchange of nuclei to produce diploid mycelium, indicating sexual mating, although clamp connections are not formed. Diploid cultures appear to be somewhat more aggressive in causing decay than haploid cultures.

Fomes igniarius does not develop extensively as a free living saprophyte. Once a tree is infected and killed, the fungus will persist on the dead

wood for a considerable period, but it does not appear to be well-adapted to perpetuation on dead fallen timber alone.

Epiphytology. The very extensive geographic range of this fungus indicates that it is tolerant of a wide variation in environmental conditions. In culture the fungus grows between temperatures of 39° and 102°F. with most vigorous development occurring between 74° and 86°. It flourishes likewise under both humid and dry conditions. The most important factors favoring its development have to do with the host tree, its advancing age, thinness of its sapwood, its proximity to other infected trees, its lack of vigor or suppression, and particularly the presence of wounds. Any factor that promotes wounds, scars, or broken branches favors infection. By the nature of the disease it is not of a type that fluctuates in severity from one year to another, but each year advances steadily and evenly in its destruction.

Control: 1. IN INDIVIDUAL ORNAMENTAL OR FRUIT TREES. Diseased trees should be removed and burned and replaced with young, vigorous trees of the longer-lived species. Broken branches and pruning and other wounds should be promptly smoothed off and protected with shellac or tree paint. The surroundings should be kept in a sanitary condition, free from dead or decaying wood.

2. IN WOODLOTS OR OTHER SMALL, MANAGED PLANTINGS. Here the removal of diseased trees and their prompt use for firewood is practical. It is not sufficient to remove the conks, as new ones will soon form. Thorough inspection is necessary to eradicate all conk-bearing trees. Fallen decaying lumber and slash from logged-off trees should be destroyed.

3. IN FORESTS. Here, because of the acreages involved, the preceding methods of control may be impractical. It is most important to follow a cutting practice which allows harvesting timber at an age when substantial growth has been obtained and decay has not yet become important. Decay is often a sign of overmaturity.

The studies of Meinecke dealing largely with *Fomes igniarius* decay of aspen illustrate a method of determining the most profitable cutting practice:

By use of an increment borer on many trees of all age classes, it is possible to determine the ages of the trees and the condition of each with regard to decay. Simple formulas indicate the amount of decayed wood and the amount of merchantable wood in board feet. Meinecke's work shows that in the type of forest studied, the total amount of timber in the forest steadily increases until the trees are about 120 years old (Fig. 32). Decay is hardly a serious factor until the age of 75 to 80 years, after which the volume of decay increases as rapidly as the volume of new wood, i.e.,

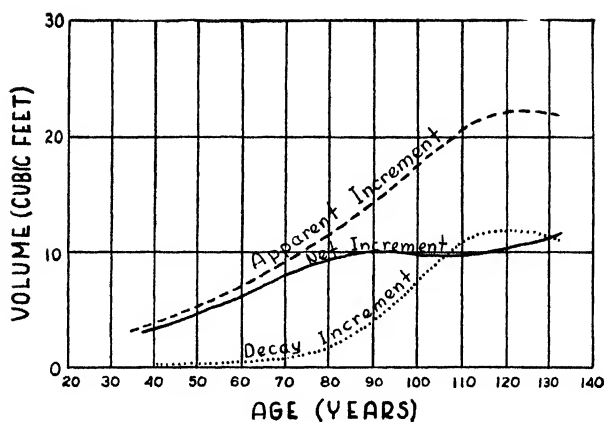


FIG. 32. Apparent, decay, and net increments of timber in an aspen stand affected principally by *Fomes igniarius*. (After Meinecke, U. S. Dep. Agr., Tech. Bull. 155.)

the net amount of merchantable wood in the forest (total wood less decayed wood) becomes constant, and there is no increase from year to year. The figure shows that at approximately 80 years there is no further annual increase in merchantable wood because the increase in decay volume (decay increment) equals the apparent increment so that there is no net increment (increase in useable timber).

From the pathologic standpoint, the desirable practice for this type of forest is, then, to cut when the trees are 80 years old or less, in order to escape serious losses from decay and get a maximum return from the investment. In a similar way the most profitable cutting cycle can be determined for each species of tree and each type of forest.

In addition to avoiding decay by following an optimal pathologic cutting practice, other means that aid in reducing decay losses include: an effective fire-control program to minimize fire wounds through which decay fungi gain access to trees; encouraging the growth of the more decay-resistant species of trees; logging contracts that require the removal of decayed trees and slash at the time the sound wood is logged off; "hot logging" or avoiding delay between cutting and utilization; sanitation of storage yards; stacking in such a way as to provide adequate ventilation of boards; proper seasoning of lumber; treatment with preservatives of wood that is to be exposed to weather and soil; and development of ways for utilizing partially decayed wood. For wood that is to be used in moist locations that are conducive to decay (fence posts and wood about the bases of buildings) preference may be given to the more durable, or decay-resistant species which include the cedars, southern cypress, redwood, Pacific yew, osage orange or Bois d'Arc, black locust, black walnut, catalpa, and red mulberry.

The less durable woods, or the more durable ones that are used in decay-favoring locations, are profitably treated with decay-retarding wood preservatives before placing in final locations. The principal chemicals used are chromated zinc chloride, zinc chloride, creosote, and 5 per cent pentachlorophenol in light oil. The zinc chlorides are used at the rate of 1 lb. in $\frac{1}{2}$ gal. water for a sapling 4 in. in diameter and 30 ft. long. A simple method is to place the freshly cut pole end in a trough of the solution for from 6 to 24 hours. Such treatment will frequently increase the life of wood by many years, and the popularity of use of treated wood is indicated by the fact that in the United States some 200,000,000 gallons of creosote and 5,000,000 lbs. of the zinc chlorides are used annually for wood preservation.

SHOESTRING ROOT ROT (*Armillaria mellea*)

History and Distribution. Shoestring root rot of many species of woody and herbaceous plants, also known as honey root rot or *Armillaria* root rot, is worldwide in distribution and important wherever it occurs. It is a constant feature of wooded areas. The causal fungus had been known as a mushroom for many years before Robert Hartig, in 1873 and 1874 proved that the underground mycelium of the mushroom, *Armillaria mellea*, is the cause of the root rot disease. An extensive bibliography of the disease has accumulated, the studies dealing primarily with the mode of infection of *A. mellea* and its control.

Importance. Shoestring root rot is a major disease of woody plants because of its destructiveness to trees, its wide host range, and its prevalence. It has been regarded as causing the death of more trees in Europe than any other parasitic agency. Piper and Fletcher give figures showing the killing by this organism of half the trees in a 1000-tree prune orchard within six years and in another orchard, one-fourth of the trees in three years. In the great forests of the Pacific Northwest shoestring root rot is a principal factor in the destruction of valuable timber, and in the Ozark region it has been regarded as one of the most serious fruit tree diseases, capable of destroying entire apple and peach orchards in two years. While the fungus primarily affects woody plants, its attack on potatoes has received attention in Australia and in the more northern potato-growing states. As it is a very common organism in oak, the most serious effects on orchards usually follow the planting of fruit trees on recently cleared oak land.

Host Plants. At least 200 species of plants are attacked by *Armillaria mellea* including conifers and deciduous forest, fruit, and shade trees, woody and herbaceous ornamentals, and vegetables. It has been called

practically omnivorous on woody plants. The most important hosts include apples, apricots, citrus fruits, olives, peaches, plums, prunes, cherries, alder, beech, birch, walnut, almond, chestnut, locust, maple, mulberry, oak, sycamore, and poplar, and of the conifers, cedars, firs, hemlocks, larch, pine, and redwood. The bush fruits, such as blackberries and raspberries, are very susceptible, as are certain ornamentals such as rhododendrons, azaleas, and boxwood. Other herbaceous hosts beside the potato include carrots, parsnips, rhubarb, dahlias, cannas, and strawberries. There is some evidence that two strains of the pathogen exist in America, one on conifers, the other important on oak and fruit trees. Of the few resistant or immune species, the pear, California black walnut, and fig may be mentioned. Myrobalan plum is resistant, giving it value as a rootstock for the more susceptible stone fruits.

Symptoms and Signs. Usually the first indication of trouble is a decline in vigor of the tree, with cessation of growth, yellowing of foliage, abnormally small leaves, and short twig growth. Trees often die quite suddenly after displaying these symptoms. In conifers and stone and citrus fruit trees, there is an exudation, often very copious, of resin or gum, especially at the tree bases. These symptoms are not always the result of shoestring root rot; similar decline may be due to drought and other causes. Positive determination of the cause of the trouble is based on the signs: rhizomorphs, mycelial fans, and fruiting bodies of the fungus (Fig. 33). The rhizomorphs are black, shiny or dull mycelial cables, $\frac{1}{25}$ inch to $\frac{1}{12}$ inch in diameter, that ramify in the soil, over the surface of roots and through the decaying wood, or spread out in a branched system, often branching at right angles, under dead bark. If dead bark is chipped away, white fans of mycelium can often be seen between bark and wood. The mycelium in infected wood is phosphorescent, causing the wood to glow in the darkness.

In fall, under wet conditions, the fruiting bodies or mushrooms appear about the bases or on the trunks of affected trees. Usually these are produced in groups or clusters and disintegrate soon after the spores are produced. The mushroom consists of a thick stipe or stem from 3 to 10 in. long, terminating in a hemispherical, umbrella-shaped cap 2 to 5 in. wide, honey-yellow and covered with scales. Below the gills there is a ring or annulus around the stipe. Careful digging will often show a rhizomorph connecting the mushroom with nearby rotted wood. The mushrooms are relished by connoisseurs as among the best of the edible fungi. The rhizomorphs are most useful in identifying the trouble, as the mushrooms are found only infrequently, and mycelial fans may be produced by fungi other than *A. mellea*.

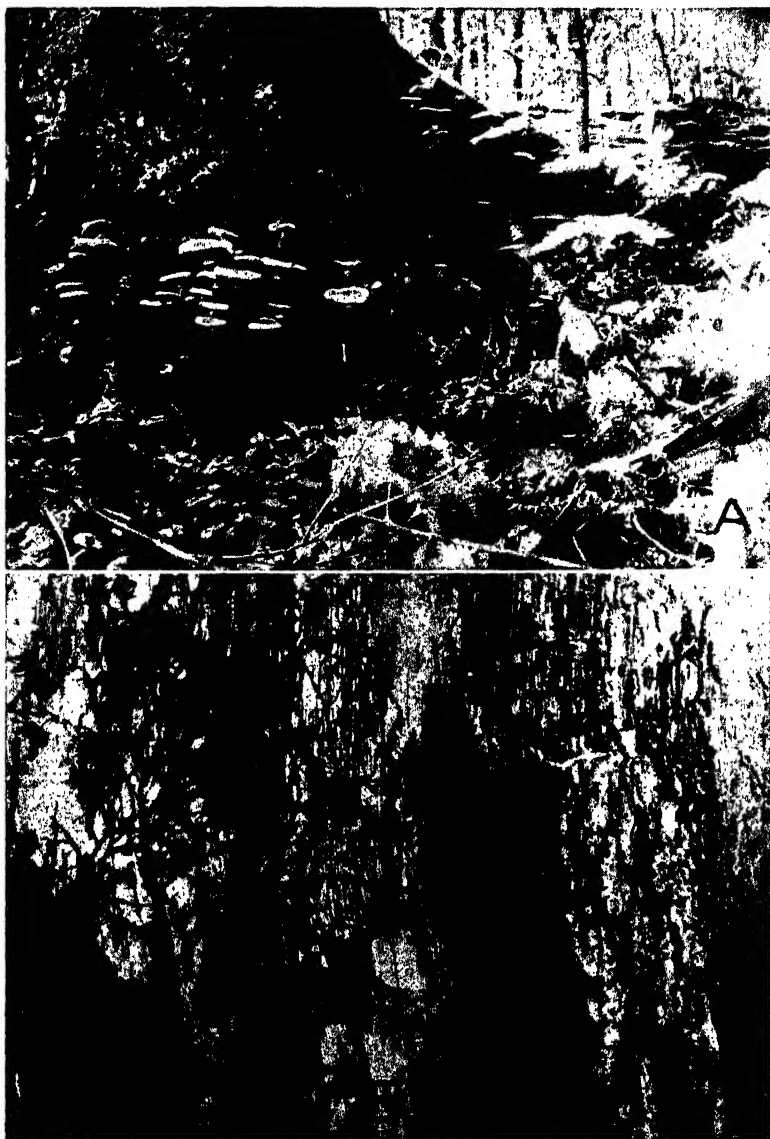


FIG. 33. (A) Base of large, dead yellow birch with many fruits of *Armillaria mellea*, showing the collar around the stem under the cap and characteristic manner of growth near the surface of the soil. (B) Shoestrings between the bark and wood of a dead tree. (Photographs, J. Franklin Collins, New England Sec. Soc. Am. Foresters, Tree Pest Leaflets.)

The decay involves both heart- and sapwood. At first the affected wood appears watersoaked, then discolored yellowish or brownish, and in final stages it is white or light yellow, marked with black zone lines, and very light and crumbly.

Etiology. *Armillaria mellea* is a gill fungus (agaric) related to the common edible mushroom of commerce. It lives very extensively as a saprophyte on decaying wood and is a common inhabitant of the forest floor and the woodpile. Two means serve for its dissemination, the wind-borne basidiospores which are produced on basidia lining the surfaces of the sporophore gills, and the rhizomorphs by which the fungus grows readily through the soil, often for considerable distances. The basidiospores are very small ($6 \times 9 \mu$), clear, elliptical, and produced in enormous numbers. In a related fungus a discharge of 40,000,000 spores per hour from a single mushroom has been recorded. These spores may germinate and invade wood under moist conditions, but the rhizomorphs appear to be the exclusive means of attacking living trees. The rhizomorphs may penetrate living, uninjured bark and wood but most frequently take advantage of wounds of any description in entering the plant. Penetration is partly through mechanical force and partly through a suberin-dissolving enzyme. Wood that is depleted of carbohydrates or wood that is previously invaded by other fungi is not attacked by *A. mellea*. Once a plant is invaded, the mycelium rapidly grows through the wood, killing cells in advance of it, digesting their components, and thus producing decay. As the roots are destroyed the water supply is cut off and the tree succumbs. In invasion of the more resistant species, the fungus enters but is walled off by secondary cork layers formed by the host, and thus prevented from extensive invasion. On the death of the host the fungus continues a saprophytic life until the wood is completely decayed.

Epiphytology. The wide geographic range of the shoestring fungus, from cold to subtropical climates and from wet forests to prairie, gives evidence of its ability to flourish under the most varied environmental conditions. While abundant fall moisture is necessary for mushroom production, the omission of their development does not interfere with the success of the pathogen which is well able to sustain itself in the form of mycelium and rhizomorphs. The condition of the host plant, on the other hand, does appear to affect the development of the disease. Devitalized trees, those suffering from drought injury, insect attack, freezing injury, or sudden exposure after cutting of adjacent trees, and trees with wounds are much more severely attacked than vigorous, sound trees. Root attack occurs at temperatures between 45° and 77°F . with optimum infection at 59° to 77° for plants that grow best in cool soil, and 50° to 64° for plants

that prefer warm soil, i.e., the least attack occurs at soil temperatures most favorable for growth of the host plant.

The disease is not one that occurs in epiphytotic proportions in certain years and becomes unimportant in others, but instead it produces uniformly serious losses each year. This is characteristic of a disease that is primarily soil-borne and exhibits a wide tolerance of environments.

Control: 1. SHADE AND ORNAMENTAL TREES. Ordinarily it is not practical to attempt to save a tree once it is obviously infected. With particularly valuable trees where the decay has not progressed to an advanced stage, promising results have been obtained by removing the soil to expose the larger roots, pruning out affected roots, treating cut ends with creosote or Bordeaux paint, and allowing aeration of the root system. One good method of removing the soil without serious root injury is to wash it out with a powerful stream of water. Sites from which diseased trees are removed may be made safe for replanting by disinfesting the soil with CS_2 or other volatile soil disinfestants. (See p. 463.)

2. ORCHARD TREES. Here the most important concern is to avoid planting an orchard of susceptible fruits on recently cleared oak land for a period of three to five years after clearing. In that time the fungus will disappear from the soil, especially if the land is used for cultivated farm crops. It has been suggested that when a tree is known to be infected or has been removed, it is practicable to surround the site with a trench 1 ft. wide and 2 ft. deep to prevent the rhizomorphs from growing across to nearby healthy trees. Infected trees should either be removed and burned or possibly treated as described above. Susceptible species should not be planted for at least three years on the site where a diseased tree has stood. Measures aimed at increasing the vigor and general health of the tree and at reducing wounding of the root system will aid in preventing losses from this disease.

3. FOREST TREES. *Armillaria mellea* is widespread in forest soils and here the treatment of individual trees is impractical. It is advisable to avoid logging injuries to tree roots. Drastic opening of a stand will injure the remaining trees by exposure and thus render them more liable to root rot damage. Thinning lightly and maintaining conditions for vigorous growth are indicated. In establishing new forest plantations, the trees, particularly conifers, should not be planted on recently cleared oak land.

4. BIOLOGICAL CONTROL. *Armillaria mellea* is a serious pest in tropical plantations and various methods of clearing and trenching have been used in attempted control. In tea culture in Africa it is customary to find stumps of old trees among the young trees and these become infested after dying and serve as reservoirs of inoculum. Wood depleted of car-

bohydrates is not invaded by *A. mellea*, and use is made of this fact by removing a ring of bark about the trunk a year before cutting the tree. Another source of inoculum is *Armillaria*-attacked woody prunings. By leaving these on the soil surface a month or more they become invaded by other fungi and then cannot serve as a substrate for *A. mellea*. This also explains why quickly-rotting woods are not usually reservoirs of *A. mellea*,

since other wood decay fungi are able to invade them more rapidly than the shoestring fungus.



FIG. 34. Mycorrhizal roots of pine seedling, showing the short, stubby, sometimes branched fungus-covered side roots.

Mycorrhizae

Many trees require the presence of symbiotic fungi in or on the roots in order to grow normally. The relationship of tree root and symbiotic fungus is known as mycorrhizal. Recent extensive tree plantings in the Great Plains for wind and water erosion control are bringing out the necessity of supplying appropriate mycorrhizal fungi in new plantings on prairie soils that normally lack these organisms. No less than 16 extensive nursery failures in various parts of the world have resulted from ignorance or disregard of the need for this symbiotic relationship. Many hundreds of species of higher plants are known to require association with mycorrhizal fungi for normal growth, including trees, grasses and other herbaceous plants, and even ferns. Absence of mycorrhiza in these cases leads to pathologic plant de-

velopment, particularly slow, stunted growth.

Types of Mycorrhizae. Mycorrhizae are of four sorts: *ectotrophic mycorrhizae*, in which symbiotic side roots are very short and are surrounded and cortically invaded by a mantle and net of fungus hyphae which serve to replace the wanting root hairs (Fig. 34); *endotrophic mycorrhizae*, in which the roots are normal outwardly but contain tangles of mycelium within the cortical cells; *ectendotrophic mycorrhizae*, in which the features of the first two types are combined, and *pseudomycorrhizae*, which

resemble mycorrhizae but are nonbeneficial or injurious invasions of roots by pathogenic soil fungi. Ectotrophic mycorrhizae are found associated with trees, while in herbaceous plants the predominating type is endotrophic.

Mycorrhizal Fungi. The fungi that participate in mycorrhizal relationships for the most part are members of the hymenomycete group of basidiomycetes although certain lower phycomycetes are sometimes involved. Many of the mushrooms and puffballs to be found in woodlands are fruiting bodies of mycorrhizal fungi, as is seen in the fact that certain species of fleshy fungi are found chiefly or only about the roots of given species of trees. Many of these relationships are quite specific, i.e., a single species or strain of fungus may be required for a given species of tree. In a few cases the mycorrhizal fungus may be a species that under some conditions is capable of severe parasitism, as is the case with *Armillaria mellea* and species of *Rhizoctonia*. Indeed, there is some evidence that the mycorrhizal relationship is the end-product of an evolutionary trend in which an earlier active parasitism has stabilized into a noninjurious and finally a beneficial relationship.

Function of Mycorrhizae. The significance of mycorrhizae has been disputed in the past, some believing them to be harmful, others beneficial, but recent experimental evidence indicates clearly that mycorrhizae are not only an aid but sometimes an indispensable aid to normal plant growth. This has been shown particularly in experiments with mycorrhizal and nonmycorrhizal trees in prairie soils, where the trees without the mycorrhizae make very poor growth and exhibit symptoms of starvation. The best evidence at present indicates that the ectotrophic mycorrhizae increase the absorbing areas of roots, permitting the plant to absorb far more phosphorus, potassium, nitrogen, and doubtless other substances, than is possible without the fungus. In addition, they offer protection against the collapse of rootlets during drought.

Control of Mycorrhizal Deficiency. Whenever trees are introduced on soil that has not supported the same species of trees in recent years, it is advisable to infest the soil with the proper mycorrhizal fungi. This may be done by interspersing the new planting with inoculated trees or soil from other localities where the mycorrhizal relationship is already established. Small amounts of soil sprinkled in the new planting will suffice. This, of course, involves the danger of bringing in harmful pathogens at the same time, but the benefits of adding the mycorrhizal fungus will ordinarily outweigh the hazards involved, especially if the inoculum comes from a nearby natural, vigorous stand or a thrifty, reasonably old plantation. Once the species of fungus required is known,

the possibility of using pure cultures for inoculation becomes apparent, and this would furnish an entirely safe means of artificially producing the desired mycorrhizal relationship.

RHIZOCTONIA DISEASE (BLACK SCURF) OF POTATOES

(*Corticium vagum*¹ = *Rhizoctonia solani*)

History and Distribution. The fungus *Corticium vagum* is one of the most widespread of all pathogenic soil fungi. In nature it is most frequently found in the imperfect stage, in which it has long been recognized under the name *Rhizoctonia solani*. In 1903, Rolfs discovered that the perfect stage was a previously known crustlike hymenomycete, *Corticium vagum*, but contrary to the best usage the name *Rhizoctonia*, referring to the stage ordinarily found, is the name most commonly used today in referring to this fungus or to the disease it causes in potatoes.

Pathologically, *R. solani* has a double interest, first as the cause of a root and stem disease of potatoes, beets, and other root crops, and second as one of the most important organisms producing damping-off of seedlings of many species of plants. At this point discussion is limited largely to the former, the subject of damping-off, as produced by numerous soil fungi, being reserved for later consideration. *Rhizoctonia solani*, on various hosts, occurs in all parts of the world, and is a factor in production wherever potatoes are grown. This is due both to its ready transmission to new areas on tubers, and to the fact that it is a normal inhabitant of many virgin soils. It was first recognized in Europe in 1858 and in America about 1900, although it doubtless had been common in both areas long before these dates. The early reports frequently confused *R. solani* with a closely related form, *R. crocorum*, the cause of violet root rot of alfalfa, beets and other crops. The *Rhizoctonia* disease is now general throughout all potato growing sections of North America, being evident everywhere, but particularly under the cooler conditions of potato culture.

Importance. The average national loss from *Rhizoctonia* is quite regularly from 2 to 3 per cent of the potato crop or about 10,000,000 bushels. Losses in the individual states range up to 15 per cent, with such important producing states as Maine, Washington, Oregon, and California occasionally reporting 8 to 10 per cent annual loss from this disease. In individual fields, losses up to 100 per cent have been experienced. The losses are of several types: (1) destroying the young sprouts before they emerge from the soil, so as to reduce the stand, (2) rotting the roots and girdling the stalks of older plants so as to shut off transport of foodstuffs,

¹ Recently renamed *Pellicularia filamentosa*, but commonly referred to in the literature under these names.

resulting in poor yields, (3) predisposing the tubers to decay before harvest, and (4) injuring the quality of the harvested crop by unsightliness, which results in a lower grade and price, or by cankering, cracking, or even rotting of the tubers. The lowering of grade is particularly costly when the potatoes are intended for certification or ordinary seed use. In fall crop potatoes, vines affected by *Rhizoctonia* are noticeably more subject to injury by light frosts than are healthy plants.

Host Plants. The list of plants attacked by *R. solani* is far too long to enumerate. Thirty years ago 165 species of plants were listed as susceptible, and doubtless many others could be added today. While most of these hosts are herbaceous dicotyledons, some of the grains and grasses, onions, and other monocotyledons, as well as several gymnosperms and equisetum are listed as susceptibles. Woody seedlings and cuttings are often destroyed by *Rhizoctonia* and occasionally the pathogen may even attack more mature woody tissues.

Symptoms and Signs. A number of striking and characteristic symptoms and signs make this disease easily recognizable. The first indication of the trouble is missing hills or hills in which the sprouts decay back, to be succeeded by secondary sprouts which in turn may decay. In later stages the plants show symptoms similar to those of drought injury, with curling of the leaves and stunting or rosetting. The stems are often decayed at or just below the soil line. This interrupts the downward flow of carbohydrates, with the result that clusters of little green or reddish aerial tubers are formed (Fig. 35A). Underground, the secondary feeding roots may be killed back extensively, to be replaced by successions of adventitious rootlets. Affected vines show a high percentage of small tubers. The best known sign is the presence on the tubers of crustlike black sclerotia, the "dirt that won't wash off." These may be few or so numerous that the greater part of the surface of the tuber may be covered by them (Fig. 35C). Other tuber symptoms sometimes encountered are a brown, deep stem-end rot, jelly-rot of the stem end, and dry lesions at the lenticels. In advanced stages of the disease, and where moisture is suitable, the perfect stage of reproduction may be seen as a white, powdery crust partly or entirely girdling the base of the potato stem, the powder consisting of large numbers of basidia and basidiospores (Fig. 35B).

Etiology. The primary inoculum may come from either of two sources—the soil, or sclerotia on the seed tubers. Under favorable conditions the sclerotia of the soil or tubers germinate by producing mycelium which is able to attack the roots and stems of the potato, initiating decay. The mycelium continues to invade the tissues, killing the cells until the characteristic root and stem rot symptoms are produced.

During this period of active parasitism, no spores are produced. Secondary sclerotia may form in the soil and these in turn may germinate to produce secondary cycles of infection, but the principal mechanism of progressive invasion is the mycelium itself. The mycelium of *Rhizoctonia solani* is easily recognizable. The hyphae are large and coarse, at first pale, later brownish, with coarse side branches that often depart from the main hypha at right angles. At the point of departure there is a slight constriction of the side branch and just out from this a cross-wall. (Fig. 129.)

In the later stages of the disease, mycelium aggregates about the stem bases in the form of a mantle or crust covered with club-shaped basidia, each bearing at its tip four basidiospores. The function of the basidiospores is obscure. They evidently play little part in dissemination of the disease during the growing season, but they may be important sources of soil infestation, returning to the soil where they germinate to form saprophytic mycelium which in turn may produce resting sclerotia.

Between crops the fungus persists either as sclerotia on tubers or in the soil, or as saprophytic mycelium in the soil. Here the mycelium can grow

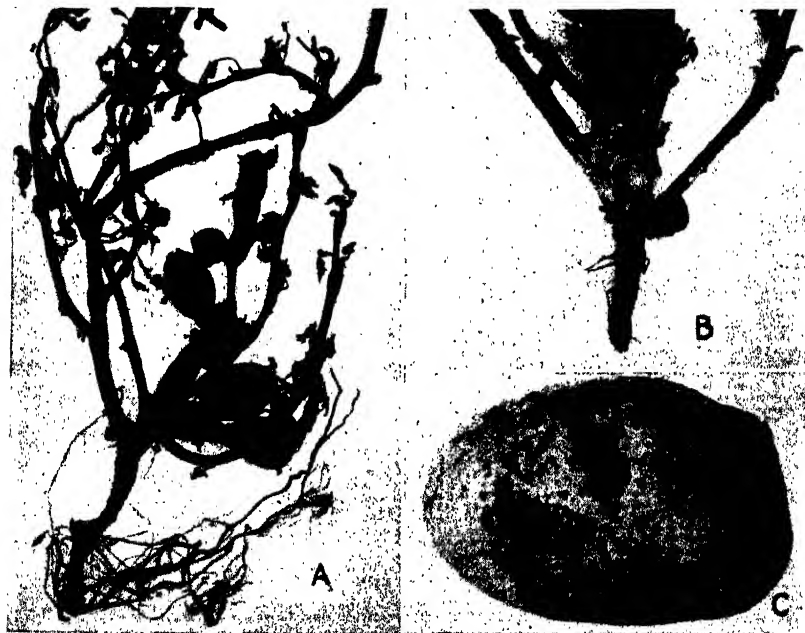


FIG. 35. *Rhizoctonia* disease (black scurf) of potato. (A) Aerial tubers are often seen on affected plants. (B) Occasionally the fruiting stage of *Corticium vagum* is seen as a white, powdery film on the lower branches. (C) The familiar "dirt that won't wash off"—black, crustlike sclerotia by means of which the fungus may persist from harvest until planting time. (Courtesy, P. E. Tilford, Ohio Agr. Exp. Sta.)

for long distances and produce sclerotia, independently of any living host. The pathogen appears to be well adapted to this soil existence.

Epiphytology. *Rhizoctonia* infection is favored by cool soil temperatures, between 41° and 64°F. Little infection results between 70°F. and 75°F., possibly because the potato sprouts develop very rapidly at these temperatures and may be able to outgrow the danger.

Ample moisture appears to favor the disease, which ordinarily is most serious in wet seasons and low wet soils.

There is some difference of opinion regarding soil reaction in its effect on *Rhizoctonia*. Textbooks commonly state that the disease is most severe on acid to neutral soils, least on alkaline soils, and partially controllable by liming.

The disease is more severe on heavy than on light soils, this perhaps being due indirectly to soil moisture in the two cases. Vigor of the plants is said to reduce their injury from *Rhizoctonia* which may relate to the ability of rapidly growing plants to grow away from the fungus.

With favorable environmental conditions the amount of damage will vary with the amount of inoculum in the soil and on the tubers, particularly the latter. As is generally true of seed- and soil-borne diseases, in contrast to primarily air-borne diseases, the *Rhizoctonia* disease does not occur in severe epiphytotics and may even disappear during some years, but produces rather constant losses each year, the only significant variation being a slow steady decrease during the past decade or two, thanks to the extended adoption of preventive measures.

Control: 1. USE OF NONINFESTED SEED TUBERS. As infested tubers are the most important source of infection, the use of noninfested tubers affords a first line of defense against *Rhizoctonia*. In securing such tubers the grower is aided both by state seed inspection service and by seed certification. Three main categories of seed are available for planting:

Certified Seed. The rules governing the inspection and sale of certified seed potatoes may limit the amount of *Rhizoctonia*, either by a direct statement or by specifying that the potatoes must meet certain U. S. standard grades which in turn limit the amount of *Rhizoctonia*. For example, according to the Nebraska certification rules (1948) Blue Tag certified potatoes must meet the requirements for U. S. No. 1 grade, which permits no more than 6 per cent of the potatoes to be materially affected by *Rhizoctonia*; Red Tag certified potatoes must meet requirements for U. S. Commercial grade, allowing up to 20 per cent *Rhizoctonia*, and White Tag and Green Tag potatoes must meet the requirements of U. S. No. 2 grade, which does not specify a *Rhizoctonia* tolerance. All of these are equally free from virus

diseases, a principal reason for certification (see p. 333), and differ chiefly in size and appearance and in tolerance of *Rhizoctonia* and other defects.

State-inspected Seed. Tubers for seed purposes usually are subject to inspection on entry into the state. The Oklahoma law, for example, permits up to 20 per cent of slight *Rhizoctonia* on seed potatoes, this being defined as "scattering *Rhizoctonia* sclerotia, $\frac{1}{2}$ in. in diameter or less in the aggregate." The Louisiana law allows up to 6 per cent of the tubers to have 5 per cent or more of their surfaces covered by *Rhizoctonia* or scab. Laws of other states are similar.

Market Potatoes. These are graded by United States standards, which affect *Rhizoctonia* as indicated under "Certified seed" above.

While these standards all seem rather tolerant of *Rhizoctonia*, it must be borne in mind that lower tolerances of *Rhizoctonia* would materially reduce the supply and raise the price of the better grades, and that other diseases, particularly virus diseases, are much more dangerous in tubers, while *Rhizoctonia* infestations can be easily removed from tubers by seed treatment.

2. SEED-TUBER TREATMENTS. While it is better to select *Rhizoctonia*-free potatoes for planting, moderately infested potatoes can be satisfactorily disinfested by tuber treatments. Several types of treatment are available:

Corrosive Sublimate Long Treatment. Soak tubers $1\frac{1}{2}$ hours in a solution of 4 oz. of corrosive sublimate (dissolved in 2 qts. of hot water) in 30 gal. of water. This is preferred in some states where it is felt to be more thorough in disinfestation than the acid mercury or organic chemical treatments.

Acid Mercury Treatment. Soak tubers 5 minutes in a solution of 6 oz. of corrosive sublimate plus 1 qt. hydrochloric acid in 25 gal. of water. Developed in Minnesota, this is the standard recommendation for *Rhizoctonia* control, with the advantage of rapidity. Acidified mercury is available commercially (e.g. Mercurnol).

Organic Treatments. Momentary dips in solutions of any of several of the newer organic seed treatment chemicals often are equal or nearly equal to the mercury treatments in *Rhizoctonia* control. Among such products are Semesan Bel, Puratized, Phygon, Tersan, Fermate, Spergon, and Arasan, each used according to the manufacturer's directions.

Yellow Oxide of Mercury Treatment. This treatment, developed in New York, has given excellent performance against *Rhizoctonia* as a momentary dip at the rate of 1 lb. of chemical to 15 gal. of water.

Chlorinated Lime Treatment. A promising treatment, originated in North Dakota, is to soak tubers in 5 per cent chlorinated lime, the length of

time varying with the temperature from 1 hour at 110°F. to 15 hours at 70°F. This loosens the sclerotia so they can be washed off entirely.

Formaldehyde Treatments. Usually these are not recommended for *Rhizoctonia* control, but are effective against potato scab. (See p. 296.)

In using the mercury treatments it must be remembered that the chemicals are poisonous, and that corrosive sublimate will erode metal, and proper precautions must be taken to avoid these hazards. In using corrosive sublimate the strength of the solution must be fortified frequently as treatment progresses. Any of the treatments should be carried out strictly according to directions as there is danger of serious tuber injury from careless or improper treatments. The treatments are for whole tubers and to avoid injury the tubers should be cut into seed pieces after treatment. On alkaline soils, where potato scab is a problem, the mercury treatments may predispose the crop to scab injury, and in this case the nonmercury organic treatments may be preferred. No treatment will be wholly effective if large sclerotia are numerous on the tubers, and such tubers should not be used for planting, even with treatment. Moreover, if the soil is heavily infested with *Rhizoctonia*, seed-tuber treatment by itself may not give effective control.

3. CULTURAL PRACTICES. A rotation in which potatoes are grown not oftener than once in three years, or preferably longer, is advised where the soil is heavily infested with *Rhizoctonia*. Crops of the grain and grass family and alfalfa are suitable for the intervening years. Potatoes should not follow sugar beets in the rotations. While this will not completely eradicate the fungus from the soil, in combination with seed-tuber treatments it will give a profitable degree of *Rhizoctonia* control. Fertilization with green manure, such as rye, stable manure, and commercial fertilizers, or a combination of these, decreases *Rhizoctonia* damage. Other cultural practices such as liming, late planting, shallow planting, and early harvesting either are of doubtful value or may be antagonistic to more important principles having to do with the optimal growing of the crop.

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Chapter 6

Diseases Caused by Ascomycetes

The ascomycetes are fungi that reproduce in two ways as a rule. In the *imperfect* stage, which is usually the stage involved in active parasitism and the production of disease, *nonsexual* spores or *conidia* are produced, often in great abundance. The conidia, sometimes called summer spores, serve for the widespread propagation of the disease during the growing season of the host. With the advent of unfavorable growing conditions, as onset of winter or hot, dry summers, when the host plant matures or goes into a state of dormancy, the fungus usually becomes saprophytic. During this saprophytic stage there appears the reproductive mechanism of the *perfect* stage, typified by *sexually produced ascospores*, borne in sack-like *asci* (plural), usually 8 ascospores to an *ascus* (singular). The ascospores are matured and discharged into the air at the onset of more favorable growing conditions; on reaching a suitable infection court they germinate, and initiate primary infections which soon result in the production of mycelia bearing *conidia*, responsible for secondary infections and the general spread of the disease.

Although many of the ascomycetes are harmless or useful saprophytes, the group includes a large number of species that are important pathogens. The principal ascomycetous pathogens are in several sub-groups, the *Sphaeriales* and kin in which the asci are contained within a flasklike *perithecium* opening by a pore; the *Perisporiales*, the perithecia of which have no opening and are called *cleistothecia*; the *Helotiales* and *Pezizales* with the asci lining the inner surface of a cuplike *apothecium*; and the *Taphrinales*, in which there is no fruiting body but where the asci are borne in exposed layers on the surfaces of host tissues. These various fruiting structures are illustrated in Fig. 36.

Similarly the conidia are produced in various ways. Usually they are borne on the tips of stalklike hyphal branches or *conidiophores*. These may be dispersed here and there over the surface of the mycelium or they may be aggregated in fruiting bodies. The latter may be simply clusters of spore-bearing hyphae that burst through the epidermis (*acervuli*), bundles of conidiophores like sheaves of wheat (*sporodochia*), or enclosed in flasklike structures called *pycnidia* from which the conidia ooze out

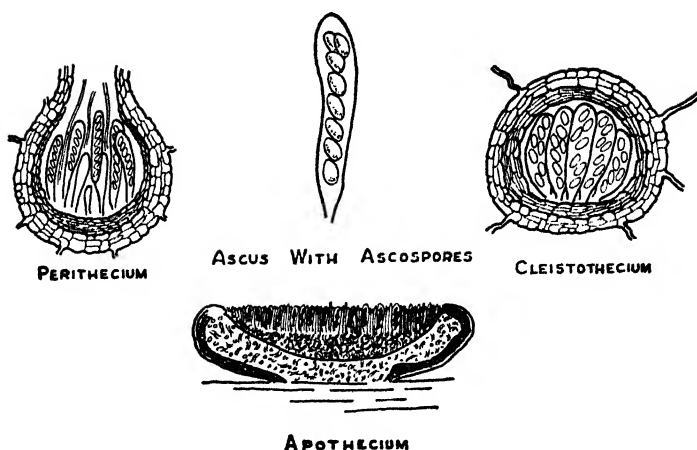


FIG. 36. Types of fruiting bodies of ascomycetes, with the ascus, common structure of them all.

through a pore. These methods of nonsexual reproduction are shown in Fig. 37.

Scab of Cereals (*Gibberella zeae*)

History and Distribution. Scab, or *Fusarium* blight, of cereals occurs in all parts of the world. In the United States it is most prevalent and destructive in the corn belt, although it is found from New York to North Dakota and California and southward to Florida and Oklahoma. The disease is one of long standing. As early as 1891 its importance in America was recognized, and since that time, besides significant losses in certain years, there have been occasional very destructive epiphytotics, as in 1919 and 1928.

Importance. The scab epiphytotic of 1919 cost American growers 80,000,000 bushels of wheat, the greatest loss occurring in Iowa—one-fourth of the crop. In other years the national losses in wheat range from 500,000 to 8,000,000 bushels annually; in addition, the disease causes

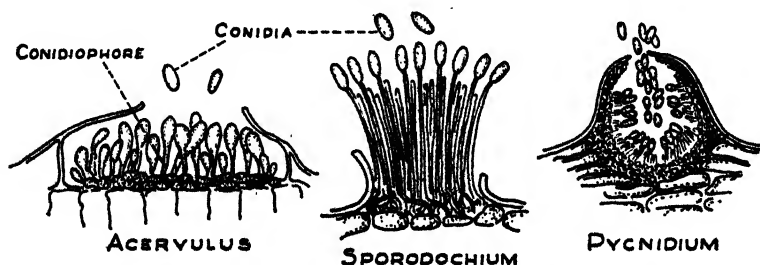


FIG. 37. Types of fruiting structures seen in asexual reproduction in the ascomycetes.

significant losses in barley, corn, and rye. The bulk of these losses are in the central states where wheat is seriously affected; in the South the disease is less prevalent, except in corn. The losses are of various types: poor stands, lowered yields through root and stalk rot, and inferior quality of the harvested grain. Scabby barley contains a poison with an emetic effect that can be tolerated by hogs only in very small amounts. Bread made from scabby rye is poisonous also.

Host Plants. Besides the cereals, wheat, barley, corn, rye, oats, spelt, and emmer, the fungus also causes a disease of clover and alfalfa, and has been recorded as a parasite of sweet potato, pokeweed, and umbelliferous plants. Wheat, barley, and corn, however, are the principal victims.

Symptoms and Signs. Cereal plants are attacked at all stages of development and the various manifestations of the disease are referred to as seedling blight, foot rot, stalk rot, and head blight or ear rot. In seedling blight, young seedlings are invaded by the fungus and sometimes totally destroyed before or soon after emergence, or so badly injured that the plants remain crippled for life. The roots of the developing plant may be destroyed progressively in the foot rot or root rot stage, producing inferior or worthless plants. In corn the scab fungus attacks ears, coming in from the ear tip, and causing a reddish decay not usually involving the whole ear. In the southern states the corn root and stalk rot stages are common; head blight is seen rarely. In head blight or ear rot, the glumes, husks, and

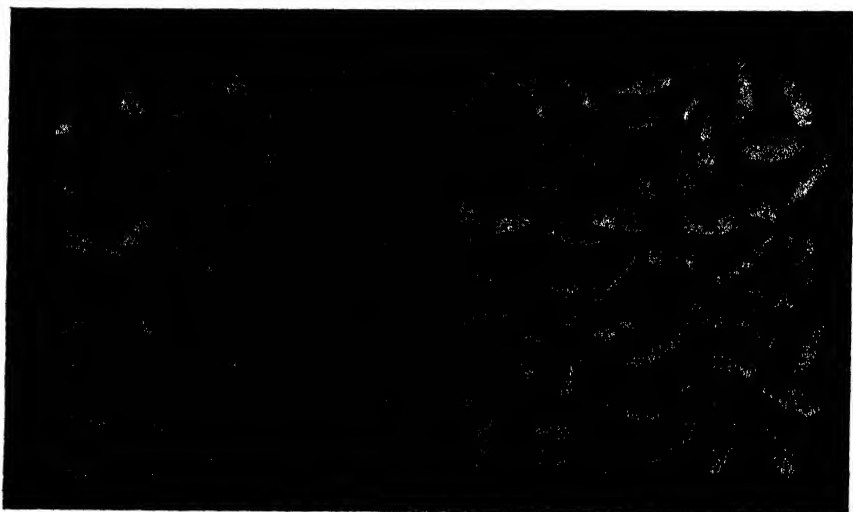


FIG. 38. Scab in wheat. (A) Healthy kernels. (B) Scab-infected kernels shriveled, light in weight, bleached, and sometimes showing a pink discoloration or orange, waxy scale of scab spores. (Courtesy, Benjamin Koehler, Ill. Agr. Exp. Sta.)

grains become overrun with white, cottony mycelium on which, in moist weather, are produced abundant salmon-colored masses of conidia. In dry weather these may dry down to form orange, waxy scales or crusts on the infected parts. The grains become invaded by the fungus in which case they are shriveled, molded, discolored, or partly rotted, often with scabby, tufted mycelium over the seed surface (Fig. 38). In small grains the affected heads are partly or entirely straw colored (Fig. 39). Late in the season or, in the South, during the following season, numerous small black bodies may be seen over the surface of affected parts, especially the glumes of small grains and corn stalks; these are the perithecia which contain the ascospores.

Etiology. The fungus causing scab is an ascomycete, *Gibberella zeae*,¹ the imperfect or conidial stage of which is *Fusarium graminearum*. Two other closely related *Fusaria*, *F. culmorum* and *F. culmorum* var. *cereale*, are associated with such similar symptoms, especially in the northern regions, that they are considered together with *G. zeae* in the etiology of scab.

Gibberella zeae overwinters in several ways: in the perithecial stage on blighted grain heads, straw, and corn stalks, as saprophytic mycelium in crop residue, as conidia on seed or in the soil, and in the case of *F. culmorum* as vegetative resting spores (*chlamydospores*).

The initial infections may be brought about in various ways. If infested seed or soil is concerned, the first evidence of disease may be the blighting of seedlings. As the fungus grows through the seedling tissues, destroying them, there are produced on the tissue surfaces large numbers of conidia. These are pink in mass, colorless under the microscope, sickle-

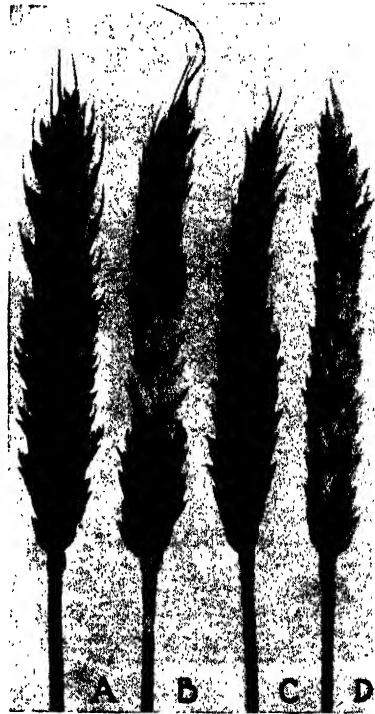


FIG. 39. Scab in wheat. (A) Healthy head. (B) Head with central part affected. (C) Head with upper half affected. (D) Entire head affected. At harvest, the kernels of affected parts will appear as in Fig. 38. (Courtesy, Benjamin Koehler, Ill. Agr. Exp. Sta.)

¹ This name is now considered correct; *G. saubinetii*, however, is the name by which the fungus is commonly referred to in the literature on this disease.

shaped, generally six-celled. They are detached easily and blown about by the wind. They may be responsible for head infections, or such infections may result from ascospores that are shot out of the overwintered perithecia. The ascospores somewhat resemble the conidia. They are spindle-shaped, slightly curved, largely three-celled, colorless, borne eight in an ascus. During the growing season there may be successive crops of conidia, producing secondary cycles of infection until entire fields are destroyed. At harvest the fungus passes into a dormant stage or continues in saprophytic activity, feeding on the residue from the affected crop.

True physiologic specialization probably does not occur in this fungus but various isolants differ markedly in their pathogenicity; some are capable of destroying nearly all inoculated plants, while others are practically nonpathogenic.

Epiphytology. Seedling blight from the scab fungus is favored by relatively dry soil, low temperatures (46° to 65° F.) in the case of corn, and higher temperatures (68° to 86° F.) in wheat. The best corn growth occurs in warm soil, and that of wheat in cool soil, hence in both cases the least seedling blight is under temperatures that are most favorable for the culture of the crop (see p. 413 and Fig. 204).

The head phase develops most extensively at temperatures of 70° to 80° F., and under moist conditions that favor germination of the conidia. Epiphytotics occur when there is an extended succession of humid, dewy, or muggy days from heading time onward. Local variations in infestation also are in large part due to methods of handling the crop, use of rotations, and planting of infested seed. Shading by weeds, high seeding rates, and lodging may increase the amount of infection.

Control: 1. **SANITATION AND ROTATION.** The residue from infested crops, stalks, straw, and chaff that harbor mycelium, conidia, and ascospores of the fungus, is the chief source of infection. Clean plowing soon after harvest, thoroughly turning under this material, goes a long way toward controlling scab. Where scab is a problem, a rotation should be practiced in which cereal crops do not directly follow one another. Corn preceding small grains is particularly conducive to scab.

2. **DISEASE-FREE SEED.** A second important safeguard is to use non-infested seed. Seed corn should be field-selected for healthy appearance, should be quickly cured, tested for freedom from germination trouble before planting, and treated with a seed disinfestant. (See p. 256.) Small grain seed should be selected from disease-free fields, lots with numerous discolored, scabby or light-weight grains should be rejected, and the seed should be thoroughly cleaned by fanning to remove light-weight, internally infected kernels, and then dust-treated as for surface-borne smuts.

3. **RESISTANT VARIETIES.** Cereal varieties differ in their susceptibility to scab but the work in breeding for scab resistance is still in an early stage. Scab-resistant inbred corns exist, but as yet have not been satisfactorily combined in hybrids for general use. In barley, the smooth-awned varieties are more susceptible than Manchuria types. Hooded varieties are very susceptible to scab. In wheat, some Turkey selections are rather scab-resistant and, in the corn belt, Illinois No. 1 and Progress wheats have largely replaced Marquis because of resistance to scab and other diseases. Success in the control of cereal rusts and smuts by breeding has focused the spotlight on scab and other diseases which have been neglected in breeding programs of the past, and an important task for cereal breeders of the future is to combine resistance to this disease with resistance to smuts and rusts in approved cereal varieties.

Apple Scab (*Venturia inaequalis*)

History and Distribution. Scab is the first ranking apple tree disease in nearly every part of the world where the crop is grown. It has attracted attention of growers and pathologists alike, particularly in North America, Europe, Australia, and South Africa. In the United States it occurs in every state, although its greatest severity is seen in the cooler sections. Scab also is a disease of long standing. It was recognized in the United States more than 100 years ago, and had been recorded in Sweden and Germany prior to that time. There is some evidence that the disease is native to parts of the world other than America, and that it was brought to this country on imported stock. Most of the early work on scab was devoted to study of the life history of the causal fungus and its control by spraying. The interest in spraying and dusting has lasted to the present as one improvement has followed another in scab control. Among the most recent developments are the use of new organic sprays, ground sprays for eradicating the overwintering stage and, by following the overwintering stage, forecasting scab outbreaks in time for spray warnings to be of service to orchardists.

Importance. Scab is the most damaging apple disease in the nation as a whole. In the 10-year period, 1928-1937, the average national loss from apple scab was 8 per cent of the crop or over 10 million bushels of apples per year. In important apple growing states the scab losses have reached very high levels during favorable years ranging from one-fourth to one-half of the crop. This is all the more striking when we consider that scab can be completely prevented by a suitable spray program and it indicates the profit that lies within reach of progressive growers.

The losses from scab are of several sorts. The leaves are affected,

reducing the vitality of the tree, and the fruit is rendered unmarketable or of poor grade because of the unsightliness of the scab lesions and because these often serve as points of entrance for decay organisms. Even with scab freedom the cost of spraying to secure this must be entered on the debit side of our account with *Venturia inaequalis*.

Host Plants. The apple scab fungus attacks only the apple and a few closely related species. The scab disease of pears is due to a similar but distinct species of *Venturia*, while scab of stone fruits is caused by a wholly unrelated fungus. Commercial apples differ considerably in their resistance or susceptibility to scab. It is not possible to give a list of susceptible and resistant varieties, because apple varieties often differ in their degree of susceptibility from one locality or year to another, possibly owing to strain differences in the fungus. Varieties that have been reported as particularly susceptible include Winesap, Rome, Virginia Beauty, Lowry, Black Twig, Ben Davis, Gano, Delicious, Stayman, Cortland, Winter Banana, King David, and Early Harvest and, as less susceptible, Baldwin (sometimes badly attacked), Jonathan, Golden Delicious, Duchess, Transparent, York Imperial, and Grimes.

Symptoms and Signs. *On the leaves* scab takes the form of olive-drab moldy spots, especially on the underside of the leaf, $\frac{1}{4}$ in. or more in diameter, without a sharp outline. Some leaf distortion may occur as the leaf puckers about the lesions. With many leaf lesions the leaf usually turns brown and drops somewhat prematurely (Fig. 40).

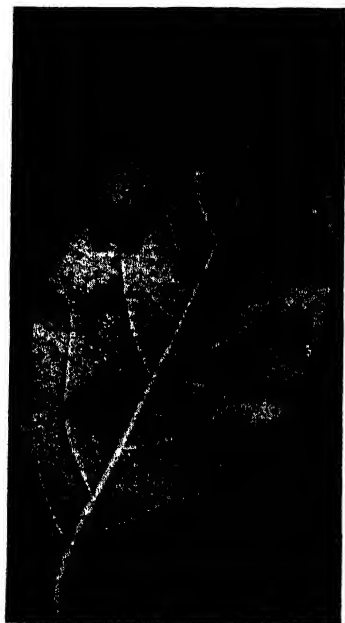


FIG. 40. Apple scab. (*Left*) Scab spots on under side of an apple leaf. (*Right*) Scab on a mature apple; note the cracks. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

On the blossoms similar olive-colored spots are seen sometimes on pedicel, calyx, and petals. Blossom infection usually is followed by the dropping of the blossoms or young fruit.

On the twigs scab lesions are uncommon but are occasionally found on one-year-old wood. They resemble those on the fruits.

On the fruits the lesions are seen first at the blossom end, but later all over the fruits. At first they are small, rounded, sharply outlined, dark, moldy spots with a white rim (Fig. 40). Later the inner dark area becomes corky and often ruptures to form a star-shaped crack. If many lesions are present, they coalesce and large areas of the fruit are cracked and split, the fruit becoming lopsided about the scabby area. Later secondary organisms, as pink and green molds, may enter through the scab lesions, causing more or less extensive decay. The scab fungus alone does not produce such decay.

Etiology. The fungus, *Venturia inaequalis*, is an ascomycete, the ascospores and asci being produced in small, dark, flask-shaped perithecia buried in the tissues of dead, overwintered infected leaves. The ascospores are brown, two-celled with one cell smaller than the other (unequal = *inaequalis*), and are borne eight in an ascus. The spores mature at about the time the apple tree is blossoming. At this time the asci are extruded through the opening (*ostiole*) of the perithecium and the spores are shot out violently. They are picked up by wind currents and, if carried to susceptible apple tissues, inaugurate primary infections. The ascospore germinates, sending out a hypha that penetrates the cuticle of the host tissue, and a layer of mycelium develops just under the cuticle. The hyphae of this mycelium branch and extend, feeding on the host tissues. In about 10 days the lesion becomes evident, the hyphae mass under the point of infection, and brown conidiophores appear on the surface, bearing at their tips one-celled, slipper-shaped brown conidia. These in turn may be carried by rain to new infection courts where they germinate and produce new infections on leaves and fruit. At the end of the growing season the infected leaves die and fall, and the fungus remains throughout the winter as a saprophytic mycelium, feeding on the dead leaf tissues until spring, at which time new perithecia and ascospores are formed.

In the past it has generally been considered that this is the only method of overwintering. Recent studies in England, however, have shown that the fungus may overwinter in the fruit bud scales and in the wood at the base of the scales. This serves to explain the formerly unaccountable value of dormant and delayed dormant sprays in contributing toward scab control.

Prior to 1901, when Clinton proved that the saprophytic perithecial

stage and the parasitic conidial stage are two phases in the life history of *Venturia inaequalis*, only the conidial stage was associated with the scab disease. As the perfect or ascospore stage was unknown, the fungus was placed in the appropriate genus of the imperfect fungi and named *Fusicladium dendriticum*. With the discovery of the sexual stage the imperfect name becomes obsolete, but it may still be found in the older literature on apple scab.

Storage Scab. In commercial storage at low temperatures, established lesions do not enlarge to a great extent, but new lesions may appear due to infections that occurred in the orchard but were not apparent at picking time. Storage scab is favored by humidity, by the higher storage temperatures, and by delay in harvesting and storing. The incubation period in storage may vary between 23 days and 6½ months. Storage scab lesions may differ considerably from field lesions, being in the form of black specks or spots up to ⅛ in. in diameter, round or irregular or star-shaped. Storage of scabbed apples proves disadvantageous also in view of the fact that the broken cuticle allows excessive transpiration, producing shriveled fruit.

Epiphytology. A study of the scab loss estimates over a series of years brings out clearly the dependence of this disease on environment and its ability to become epiphytotic under suitable weather conditions. The discharge of ascospores is most favored by temperatures from 50° to 54°F. accompanied by rains, although they can be discharged between 33° and 86°F. The release of ascospores may continue over a period of 2 to 3 months. The ascospores germinate and infect at temperatures from 41° to 79°F. Infection, primary or secondary, can occur only when there is a film of moisture on the tree. Cool rains occurring at any time after primary infection favor secondary spread of the disease, hence it will be most prevalent during cool moist seasons and inhibited by either dryness or temperatures above 80°F. These facts explain both the greater severity of the disease in northern regions or at high altitudes in southern regions, and its annual variations in destructiveness.

Other factors influencing the disease are prevalence of infected leaves from previous crops, susceptibility of apple varieties, rank growth in poorly pruned trees, low, moist locations, excessive nitrogenous fertilization, and the efficiency of the spray program.

Control: 1. **SPRAY SCHEDULE.** A suitable spray schedule has long been recognized as the main essential for scab control. The spray materials, number of applications, time of applications, and equipment vary according to scab severity, size and location of the orchard, and personal preferences. A complete scab spray schedule may include as many as ten

applications, but rarely would all of these be needed. Unless the scab hazard is great, two or three applications will give practical control. Heald considers the calyx spray as most important, accounting for 40 per cent control, the sprays preceding it giving another 40 per cent control, while the later sprays have 20 per cent value. The dormant eradicant spray reduces but does not eliminate the need for later sprays. The last two cover sprays are not always needed for scab control, but are useful against bitter rot, black rot, blotch, and other diseases and insects.

Spray Materials. In the past the backbone of the scab spray program has been lime-sulfur for the early sprays and Bordeaux mixture for the later ones. Bordeaux mixture has a tendency to burn the leaves or russet the fruit in cool weather and lime-sulfur is likely to burn the foliage in hot weather. Recently there has been a tendency away from use of these materials, preference being shown for the milder sulfurs and the newer organic fungicides. The chemical formulas of these, and their several uses and limitations, are given in Chapter 20.

Fermate is generally recognized as a good substitute for sulfur without danger of burning the foliage. It can safely be mixed with sulfur, lead arsenate, nicotine, DDT, zinc-lime safener, and summer oil, but may be injurious if combined with lime or copper sprays. Most sprays are protectants only; they prevent spread of disease but do not kill spores at their source. Fermate has the added advantage of inactivating or "burning out" scab lesions, permitting greater latitude in spray timing without loss. It may be used for the cover sprays, 1 lb. per 100 gal., with lead arsenate, 4 lbs., and summer oil, 2 qts.

In some states, though not in all, Dithane plus zinc sulfate and lime, and Isothan Q15 have performed very well in scab control, particularly for the earlier sprays. Puratized is a promising new organic scab spray which is both eradicant and protectant, very safe, and leaves no visible residue. It contains mercury, however, and it cannot be used after the first cover application for fear that the mercury residue on fruit might not meet the standards of the Pure Food and Drug Act. Phygon is another of the newer organic fungicides which gives good protection against scab but has a tendency to spot the fruit or yellow the leaves of some apple varieties.

Applications. Spray schedules for apple scab control differ from one state to another, and change from year to year with added experience and the testing of new materials and schedules. The following is a representative schedule, but in order to obtain the latest recommendations under their particular local conditions growers should at all times keep in touch with specialists in their agricultural experiment station, either directly or through county agents or vocational agriculture instructors.

- a. *Dormant spray*, in early spring when the trees are dormant. This is often an oil spray, for insect control, but serves as a good eradicator spray destroying the scab fungus in dead leaves if a formula like the following is used: oil emulsion or miscible oil, 3 gal., plus either Dinitro or DN, 1 to 2 lbs., or Elgetol 1 to 3 qts. in 100 gal. of water.
- b. *Delayed dormant spray*, at the time the blossom buds show $\frac{1}{4}$ in. green, consisting of liquid lime-sulfur, 2 gal. in 100 gal. of water. This is sometimes omitted if the dormant spray has been applied.
- c. *Prepink spray*, when the pink of the blossom buds can first be seen, consisting of $1\frac{1}{2}$ to 2 gal. of liquid lime-sulfur, or 8 lbs. of dry lime-sulfur, or 12 lbs. of flotation sulfur per 100 gal. of spray.
- d. *Pink spray*, when the blossom buds are enlarged and beginning to open, but before full bloom, consisting of any of the following: lime-sulfur, $1\frac{1}{2}$ to 2 gal. or 8 lbs.; flotation sulfur paste, 12 to 16 lbs.; wettable sulfur, 8 lbs.; Fermate, $\frac{1}{2}$ lb. plus either wettable sulfur, 3 lbs. or flotation sulfur paste, 6 lbs.; each in 100 gal. of water.
- e. *Blossom spray*, used if the blooming period is prolonged by cool damp weather, consisting of flotation sulfur paste, 10 lbs., or wettable sulfur, 5 lbs., or Fermate and sulfur as in the pink spray.
- f. *Calyx, petal fall, or shuck spray*, just after the majority of blossoms have fallen, but before the shucks have closed over the fruits, consisting of lead arsenate, 3 lbs., plus hydrated lime, 3 lbs., for insect control, plus wettable sulfur, 6 lbs., or flotation sulfur paste, 8 to 12 lbs., or Fermate and sulfur as in the pink spray.
- g. *First cover spray*, 8 to 10 days after calyx spray, consisting of 3 to 4 lbs. of lead arsenate plus 3 to 4 lbs. of hydrated lime, to which may be added $\frac{1}{2}$ lb. of DDT for insect control, plus flotation sulfur paste, 8 to 12 lbs., or wettable sulfur, 6 lbs., or lime-sulfur, 6 qts.
- h. *Second cover spray*, 10 days after first cover spray, of 2-4-100, or 1-5-100 Bordeaux, or $\frac{1}{2}$ -2-100 zinc-lime, plus lead arsenate, 3 to 4 lbs.
- i. *Third cover spray*, 1 to 2 weeks after second cover spray, consisting of the same materials as in the second cover spray.
- j. *Fourth cover spray*, 10 days after third cover spray, consisting of the same materials, or using 6 lbs. of flotation type sulfur for the fungicide.

Efficiency of Spraying. To be effective, spraying must be thorough, covering (but not drenching) all foliage and fruit (Fig. 222). To facilitate efficient spraying, large orchards should be so arranged that trees bloom-

ing at the same time will be together in blocks. Unless the spraying is properly timed, and an appropriate material thoroughly applied at the right concentration, unsatisfactory results or injury will follow.

Spray Warnings. In the spring it is possible to determine the most effective time for spraying by examination of the fallen leaves on which the scab organism has overwintered. The results are broadcast to growers in the form of spray warnings.

Combined-purpose Sprays. Scab is rarely if ever the only reason for an apple spray program. Other apple diseases, such as blotch, bitter rot, black rot, and insect pests such as the San José and other scales, the curculio, codling moth, canker worms, aphids, and leaf hoppers, call for regular control each year by spraying with fungicides, arsenicals, contact insecticides, or oil sprays. In this regard each locality, each orchard, and each season presents its own particular problems to be solved by the grower with the help of agricultural advisors, making use of compatible spray mixtures designed to accomplish the most in yield improvement at the least expense.

Hazards of Spraying. Spraying and dusting are accompanied by certain dangers, such as injury to the tree, poisoning of fruit, injury to the soil, and poisoning of beneficial insects. These will be considered later.

2. SUPPLEMENTARY CONTROL PRACTICES. Although spraying is the major factor in scab control it is not the only one to consider. Spraying will be least risky and most effective if it is combined with other precautionary measures, such as diminishing the source of inoculum by disking under or burning fallen leaves, good pruning to produce a well-aerated tree and, other things being equal, showing a preference for the less susceptible apple varieties.

Apple Black Rot (*Physalospora obtusa*)

Black rot is another of the leading apple diseases east of the Rocky Mountains. The causal organism, which occurs also on peach, quince, currant, pear, and other woody species, attacks leaves, twigs, and fruits. On the leaves, where the disease is sometimes called frog-eye leaf spot, the mature lesions have a concentric ringed appearance. On the twigs ("New York apple tree canker") the canker either takes the form of a considerable expanse of roughened bark, or the wood will be killed, the bark cracked, and the limb girdled. The fruit rot is primarily one of mature fruit, a rounded, very dark, often zonate lesion that extends into the flesh, producing a dark decay that eventually consumes the entire fruit, converting it into a shriveled, black, shiny mummy (Fig. 41). Much of the loss from this disease comes from rotting of the fruit on the ground. Black,

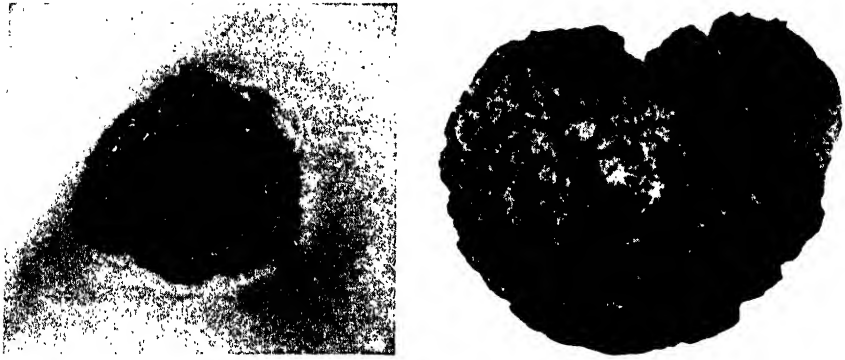


FIG. 41. Black rot of apple. (*Left*) Black rot lesion on fruit, enlarged to show the individual pycnidia. (*Right*) Mummified apple, the overwintering stage of the black rot fungus.

domelike fungus fruiting bodies (pycnidia) are found on stem and fruit lesions (Fig. 41), but the fungus rarely produces spores on leaves. The black rot fungus makes little use of the ascosporic stage in its life history, although perithecia with ascospores are regularly found in the wood cankers. Normally it overwinters as a saprophyte in the form of mycelium or as conidia in the mummified fallen fruit and in cankers. Dead trees and limbs killed by fire blight or other causes are particularly dangerous sources of spring infection. In the spring the conidia are released from their flask-like pycnidia in tiny tendrils. They are borne to infection courts by wind, splashing rains, or insects and, finding an injury, such as a mechanical bruise or insect wound, the germ tubes penetrate and begin a series of infection cycles. The fungus cannot infect uninjured tissues. Control of black rot requires the use of a spray schedule similar to that for apple scab but including only the applications from petal fall onward. Of the newer materials Fermate has shown particular promise in black rot control. Losses from the disease are reduced also by avoidance of bruising in handling fruit, storage between 31° and 34°F., pruning or tree surgery to remove the cankers, and orchard sanitation, disposing of mummies by plowing under or burning, and burning of pruned wood.

Apple Bitter Rot (*Glomerella cingulata*)

This is a late-season disease of the fruit, often very destructive in central and southern states. It is a blow to the orchardist who, having been successful earlier in the season in controlling scab and codling moth, sees the greater part of a promising crop destroyed by bitter rot.

The bitter rot fungus causes disease on other types of woody plants but this has little bearing on its attack on apple. Affected apples first show light

brown, circular spots which gradually enlarge, overlying rotted flesh that extends inward to the core. The flesh often has a bitter taste. On the surface of the lesions, which become concave, are circles of pink, later dark, clusters of conidiophores with conidia (Fig. 42). These are exuded in a sticky mass and are not wind-borne, but can be splashed about by rain or carried to other fruit by insects, especially flies. Eventually, affected apples become dry, shriveled mummies, furnishing a means by which the fungus overwinters.

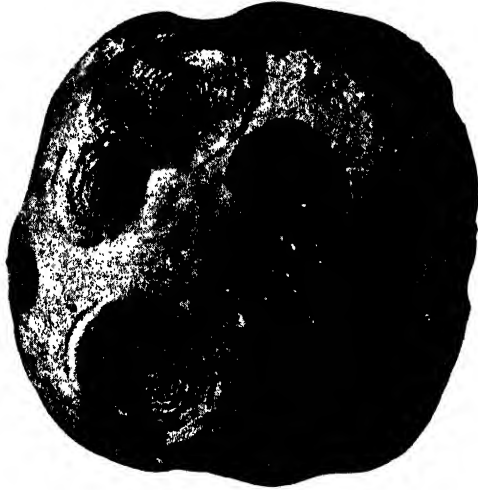


FIG. 42. Apple fruit showing lesions of bitter rot with rings of fruiting pustules (acervuli). This is another of the common apple diseases, controllable by spraying. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

Glomerella cingulata also produces roughened, oval cankers on the older limbs. These are another important source of infection in western apple sections but not in the East. An ascospore stage occurs on cankers and mummies, but this is not of great significance in the disease cycle, most primary infections in the spring resulting from conidia produced on cankers or mummies.

Apple varieties vary from highly susceptible to resistant toward bitter rot, and some varieties, such as Yellow Newtown, may be very resistant to the canker phase but highly susceptible to fruit rotting. Other susceptible varieties include Northwestern Greening, Rhode Island Greening, Stark, Ben Davis, Golden Delicious, Grimes Golden, Jonathan, Missouri Pippin, and Northern Spy, while among the more resistant varieties are

Arkansas, Baldwin, Delicious, Rome Beauty, Stayman Winesap, Starking, York Imperial, Transparent, Wealthy, and Winesap.

Control is chiefly through late season spraying but may be reinforced by knocking off and destroying the mummies, prompt removal of the first affected fruit, and removal and burning of cankers during the winter pruning. The second to fourth and even later cover sprays are most effective against bitter rot, and where this disease is a problem, Bordeaux 4-6-100 is a standard spray material. Several of the new organic fungicides are promising for bitter rot control, including Puratized, Fermate, Phygon, and zinc dimethyl dithiocarbamate (Methasan, Zerlate, Zimate, etc.).



FIG. 43. Blister canker on apple branch.

Apple Blister Canker (*Nummularia discreta*)

In Kansas, Missouri, Oklahoma, and Nebraska millions of apple trees have been killed by blister canker, which is a major apple tree disease also in the corn belt and east to New York and Virginia. The disease attacks only the wood, affecting both large and small limbs. In later stages the cankers are easily recognized as dead areas, mottled with living wood, 3 ft. or less in length, shedding the bark, and exposing numerous round, nailheadlike fungus cushions (stromata), giving the wood a blistered appearance (Fig. 43). The causal fungus, *Nummularia discreta*, is an ascomycetous wound parasite, entering through branch stubs or bark injuries, destroying the wood, and producing honey-colored conidia and later ascospores from flask-shaped perithecia imbedded in the stromata. Both conidia and ascospores appear to be involved in dissemination of the disease. Control depends almost entirely on the early detection, pruning out, and burning of cankered wood.

Grape Black Rot (*Guignardia bidwellii*)

Black rot is the most important disease of grapes east of the Rocky Mountains, causing regular losses that at times involve 30 per cent or more of grape crops in individual states. It involves all green parts of the vine. On the leaves it produces reddish-brown dead spots sprinkled with tiny

fungus fruiting bodies (pycnidia). When fruits are about half grown the disease appears as a pale spot, soon turning brown, involving the entire berry, which shrivels, becoming a black, dry mummy that will shatter off or remain in the cluster. Half to all of the grapes in a cluster will be so affected (Fig. 44). Some varieties (e.g., the Labrusca group) show leaf resistance and fruit susceptibility, and others (e.g., the Scuppernong group) show the reverse.

Guignardia bidwellii is an ascomycete, reproducing by ovoid conidia and threadlike microconidia formed within flask-shaped pycnidia on the leaves and mummies in summer and fall and in the following spring, and



FIG. 44. Grape black rot. Many of the fruits have fallen, and those remaining show various degrees of rot, from small circular spots to shriveled mummies.

by ascospores produced from stromata on the mummies that in the spring become converted into spore-bearing perithecia. Control by spraying is required in nearly every vineyard of susceptible grape varieties. In addition, the mummies should be destroyed at picking time, or at least worked under the soil in the spring. A number of grape varieties are quite highly resistant to black rot, such as Clinton, Delaware, Elvira, Ives, and Missouri Riesling.

The black rot spray schedule requires 3 to 5 applications of 4–6–100 Bordeaux mixture with a good spreader-sticker, beginning just before the vines blossom. In addition, a dormant eradicant application of Elgetol to the ground has sometimes been helpful in black rot control. Fermate

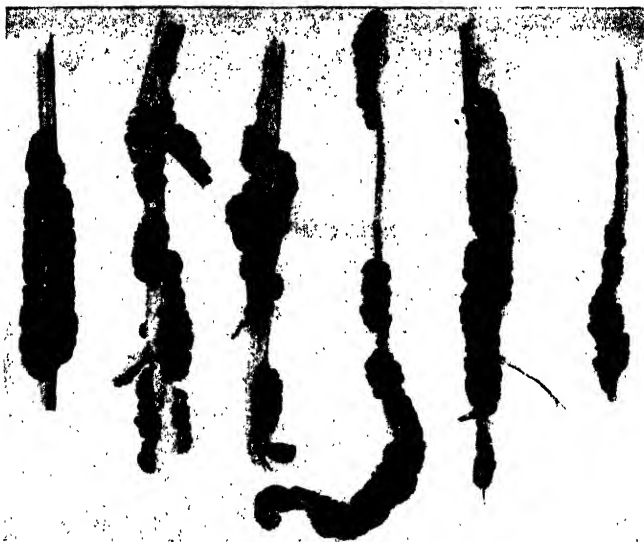


FIG. 45. Black knot of plum.

appears to be a very good substitute for Bordeaux mixture in black rot sprays.

Black Knot of Plums and Cherries (*Dibotryon morbosum*)

Black knot is a native disease of plum and cherry twigs occurring wherever these crops are grown in America, sometimes producing disastrous losses. It is easily recognized as black, rough, cylindrical, or spindle-shaped enlargements of the twigs usually two to four times the thickness of the normal twig and several inches long (Fig. 45). The twigs commonly die back to the knots. In the summer the knots become velvety with a layer of conidia, and later turn black and are covered with innumerable flasklike perithecia that liberate ascospores in the spring. Both types of spores are involved in spread of the disease. Control depends mainly on pruning out of the knots 3 to 4 in. below the swelling. This is best done in winter or very early spring when the knots are most obvious and before ascospores are liberated. Wild affected plums near the orchard should be pruned or eradicated. A delayed dormant spray of lime sulfur, 1:8, or Bordeaux 6-12-100 spray with 3 per cent emulsified oil has given good results in controlling black knot.

Elm Leaf Spot (*Gnomonia ulmea*)

Our commonest leaf disease of American and some of the Asiatic elms is the black leaf spot caused by the ascomycete, *Gnomonia ulmea*. The

spots are in the form of black raised fungus masses, partly covered or surrounded by white epidermis (Fig. 46). They may be very numerous on the leaves, in which case early defoliation results. In severe cases the twig terminals may be attacked and killed, producing a condition resembling fire blight of apple and pear, with a creamy exudate of spores. Conidia are produced on the lesions during the summer, and ascospores in perithecia are formed on the fallen, dead leaves in the spring. Young dormant elm trees brought into the greenhouse in midwinter have developed badly blighted young shoots at a time when ascospores from fallen leaves were not present, indicating overwintering of the disease in the dormant buds as well as in dead leaves. Few data on control are available. Control is required only during very wet seasons. It is suggested that the fallen leaves be raked up and burned and that the tree be protected with a dormant spray of lime-sulfur, 1:8, and with applications of Bordeaux mixture at the time the leaves are unfolding and again when they are fully expanded. Often a stomach insecticide for chewing insects is desired in the same spray, reducing the cost factor for leaf spot alone.



FIG. 46. Elm leaf spot. A severe attack on young shoots of elm, showing the appearance of the leaf lesions and killing back of the growing shoot.

Sycamore Anthracnose (*Gnomonia veneta*)

This disease is universally present on the sycamore and also occurs on oak. It is the most common and destructive disease of the sycamore. The causal fungus, an ascomycete, *Gnomonia veneta*, which is closely related to the elm leaf spot organism, attacks leaves, causing extensive dead blotches finally involving the entire leaf. The lesions are easily confused with drought or frost injury (Fig. 47). In moist seasons all the terminal



FIG. 47. Sycamore anthracnose, showing blighting of leaves and twigs.

shoots of large trees may be blighted and killed back. Cream-colored masses of conidia are exuded along the veins during wet weather. Cankers also are formed on the smaller branches and twigs, often killing back the branchlets. These bear conspicuous pimple-like spore masses in moist weather. Perithecia, containing ascospores, are produced in the spring on overwintered infected leaves. The spring infections arise either from conidia emerging from infected twigs, or from ascospores. Under damp conditions these initiate numerous secondary cycles of infection. The loss of leaves and particularly the dieback of twigs and branches may severely weaken the trees with carryover effects the next season.

The leaf infections can be controlled by a dormant application of lime-sulfur (1-9), followed by thorough applications of Bordeaux mixture (4-4-50 or 5-5-50) beginning just after the buds burst and before the first leaves are half grown, coupled with burning of fallen leaves, and pruning of dead twigs, so far as this is practicable. Failures in controlling the disease by spraying, sometimes reported, may be due to heavy twig infection, and in such cases it may be necessary to prune thoroughly and continue the spraying for two or more seasons before the disease is well under control.

Red Rot of Sugar Cane (*Physalospora tucumanensis*)

A general yield decline with repeated crop failures led to virtual bankruptcy of the Louisiana sugar industry in 1923-1926. This was due princi-

pally to successive attack by three diseases—mosaic, red rot, and root rot—to all of which the older commercial cane varieties were very susceptible. The development of high yielding cane varieties possessing resistance to these diseases has been one of the great accomplishments of modern plant breeding.

In the 1930's, red rot led to the complete failure of one of the leading commercial varieties, P.O.J. 213. The disease attacks the stalks, reddening or rotting the interior, and inverting sucrose to less desirable sugars. The midribs of affected leaves show as dark streaks, producing spores which serve to infect the stalks. Both leaf and stalk infections are favored by insect injuries, borer holes being the common point of attack on the stalks. As a result of the attack the stands of cane are reduced, soon becoming unprofitable. The fungus persists from one season to the next in infected plants, not in soil. The red rot fungus, which occurs in several strains of different appearance and virulence, has long been known as an imperfect fungus under the name *Colletotrichum falcatum*. Recently, however, an ascospore stage has been discovered but this may not have much significance in the pathology of the organism.

Control of red rot involves plantation sanitation and the use of resistant varieties. Desirable sanitary practices include the destruction of infected trash and the use of disease-free seed pieces grown in well-managed seed plots. Summer planting of those varieties that are adapted to this procedure enables plants to get a good start and escape the disease. Available resistant varieties still leave something to be desired, but are constantly being improved.

Iris Leaf Spot (*Didymellina macrospora*)

This is the commonest disease of varieties of the German iris group, rivaled in importance only by the bacterial rhizome rot of iris. It devitalizes the plants and impairs their appearance. The disease occurs on

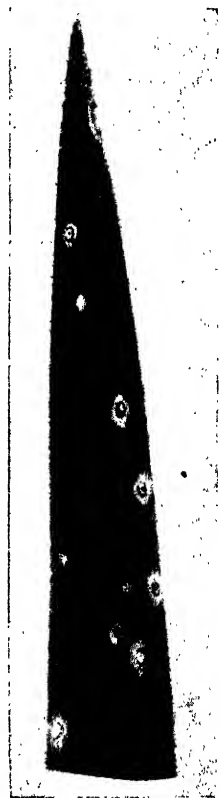


FIG. 48. Iris leaf spot, showing an early stage in the development of the necrotic spots with pale halos. In later stages the leaf dies back from the tip. (Courtesy, P. P. Pirone, N. J. Agr. Exp. Sta.)

the leaves, especially the upper portions. The spots at first are minute, brown, with watersoaked margins, later slowly enlarging, becoming yellow, then brown and dry (Fig. 48). The leaves die prematurely and in severe cases the entire plant is killed. Iris leaf spot is due to an ascomycete, *Didymellina macrospora*, that forms dark tufts of conidiophores and conidia on the surface of the lesions during the summer, and perithecia containing ascospores in overwintered leaves. The conidia are the means of rapid summer spread. The ascospores are responsible for primary infections in the spring. A practical control is obtained usually by gathering and burning the diseased leaves in the fall. Good results also follow applications of Bordeaux mixture 4-4-50 or flotation sulfur 4-50 with a good spreader-sticker. A long list of iris varieties classified according to susceptibility is given in the *Florists' Review*, 80(2072): 24-25, (1937).

Sweet Potato Black Rot (*Endoconidiophora fimbriata*)

Black rot causes greater sweet potato losses than any other sweet potato disease with the possible exception of stem rot or wilt, and is coextensive with the crop. Although most destructive in storage, it is also a seedbed and field disease. On the potatoes it appears as rounded, blackish spots extending into the vascular ring or sometimes deeper. If such roots are bedded the sprouts are often sickly, with black cankers below ground, or they are killed entirely (Fig. 49). When affected sprouts are set in the field there is more or less root decay although the plants may survive and produce a fair to good yield. Three types of spores are produced freely on infected tissues: rodlike hyaline conidia; thick-walled, ovoid, brown conidia; and ascospores, the latter borne in perithecia with extremely long necks. The rodlike conidia are produced inside tubelike conidiophores from which they are extruded endwise through the open tip of the conidiophore. Conidia of this type are called endoconidia. (See Fig. 83.) All types of spores are instrumental in spread both in the field and in storage, while the brown conidia appear most resistant. Spread of the spores and the disease in the field is aided by the activities of the sweet potato weevil. Washing harvested potatoes before storing is a dangerous practice, as this spreads black rot by means of spores in the wash water.

Control of black rot requires selection for propagation, preferably in the field, of sound roots from healthy vines, disinfestation of the roots before bedding, use of new or sterilized soil in the hotbed, and a three- or four-year rotation of sweet potatoes with any other crops. For mother root treatment before bedding it has been customary in the past to use mercuric chloride (1:1000), an organic mercury dip, or borax solution. These are effective in control, but sometimes injure the roots. Good black rot con-

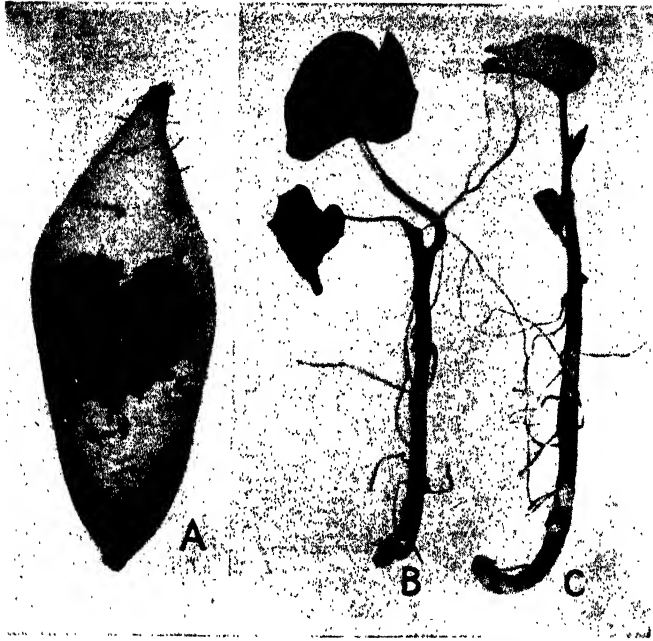


FIG. 49. Black rot of sweet potato. (A) A potato from the field at digging time, showing the slightly sunken spot with a somewhat circular outline, characteristic of the disease. (B and C) Two sweet potato plants pulled from a commercial seed bed, B showing that infection had taken place from the growth of the parasite from the potato to the stem, C from soil at a point some distance from the potato. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

trol without root injury has been reported from using as root dips the newer organic fungicides, Spergon, Phygon, Fermate, Zerlate, Tersan, Puratized, Dithane, and Isothan Q15. No commercial varieties of sweet potatoes are resistant to black rot but progress is being made in developing resistant varieties by breeding.

Dutch Elm Disease (*Ceratostomella ulmi*)

The Dutch elm disease was first observed in northern Europe in 1918, and by 1921 it had become epiphytotic and disastrous to European elms. It was brought to America on elm wood from Europe, and found in 1930 in Ohio. It appeared in New Jersey and nearby states in 1933, since when it has destroyed many thousands of trees in that area. The causal fungus, *Ceratostomella ulmi*, is a wound parasite that invades the water-conducting tissues, producing prompt or delayed wilting and death of trees of all ages (Fig. 50). Wood of affected trunks and branches shows a dark xylem ring (Fig. 51), although other diseases present a similar symptom



FIG. 50. An American elm dying from the Dutch elm disease and showing extreme wilting and partial defoliation. Such trees are a menace to nearby healthy trees and should be promptly felled and burned. (Photograph, N. Y. Agr. Extension Serv.)

and culturing is necessary for diagnosis. Conidia are produced in sheaf-like fascicles (coremia), especially in insect tunnels, and the spores often bud in the fashion of yeasts. Although rare or absent in nature, an ascospore stage has been produced experimentally by crossing + and - strains of the fungus on sterilized elm twigs. The perithecia are long-necked as in the sweet potato *Ceratostomella*. The fungus is transmitted principally by bark beetles (*Scolytus* species).

During the first years of its invasion of America the Dutch elm disease was combated by an extensive program involving the locating and eradication of diseased trees, Federal, State, and Municipal authorities co-operating. In this way the disease was very nearly eradicated from Ohio

and for a time it seemed that this might be accomplished also in the affected areas of the Eastern seaboard. But despite eradication of many thousands of trees, it appears that Nature has won the battle, that we cannot wipe out the disease entirely but must learn to live with it and keep its destructiveness at the lowest possible level.

It has been shown that valuable elms in towns and cities can be protected by maintaining a zone of disease-free elms around each locality. This can be done by prompt detection and removal of affected trees. Given a disease-free zone of 1000 ft. width, the chance of infection of trees protected by the zone is only 1 in 10,000.

When elms are killed by other diseases such as the virus disease, phloem necrosis, breeding places are provided for multiplication of the beetles that transmit the Dutch elm disease. This makes the problem a broad, complex one, involving a general health and pest control program for elms, with eradication of trees that are insect breeding places or sources of disease. Progress has been made in controlling the Dutch elm disease by injecting certain chemicals into the wood (see p. 490), and the outlook for developing desirable elms that are resistant to the Dutch elm disease looks promising.



FIG. 51. Cross section of an elm branch affected with the Dutch elm disease, showing brownish discolorations in the sapwood. (Photograph, Dep. Plant Pathology, Cornell Univ. Agr. Exp. Sta.)

Chestnut Blight (*Endothia parasitica*)

Chestnut blight, a disease introduced from Asia, first found in New York in 1904, and epiphytotic from Massachusetts to Virginia by 1908, has in succeeding years practically eliminated this valuable tree from American forests. The causal fungus, an ascomycete, *Endothia parasitica*, is a wound parasite that produces large, sunken cankers on branches and limbs, killing both individual limbs and ultimately the entire tree. The fungus fruits early and abundantly in crevices of the broken bark over the cankers, first producing conidia which exude in tendrils from numerous reddish pycnidia, and later ascospores from perithecia embedded in orange masses of fungus tissue (stromata). The ascospores are readily spread by wind—their main agency of dissemination—while the sticky conidia are carried largely by insects and birds. Tawny fans of mycelium are seen under the affected bark. Insect injuries commonly serve as infection courts.

After the tree is killed the fungus continues for about one year as a saprophyte, still sporulating. Although many control measures were tried, all failed, and the disease has run its course until the host species has been practically wiped out. It gives us a tragic lesson in the effect of an introduced pest on a susceptible native crop.

As time goes on, it is less and less likely that the chestnut can ever regain its former place in American forests. Long years of laborious breeding and selection, however, particularly using oriental chestnuts as sources of resistance, lend some encouragement to the view that eventually there will be developed desirable types of blight-resistant chestnuts for home and orchard planting.

✓ **Ergot of Grains and Grasses** (*Claviceps purpurea*)

History and Distribution. Since the time of the Caesars, long before its recognition as a fungus disease, ergot has been known as the cause of human pestilence, the "holy fire" of the Middle Ages. As the disease is most prevalent in rye, the universal bread grain of Europe, we read in European literature of many epidemics of human suffering and terrible death from the gangrenous poisoning produced by mixtures of the ergot sclerotia in bread grain. It was not until 1842 that L  veill   in France first discovered that the black ergot horns are the product of fungus disease, and a few years later the scientific farmer, Julius K  hn, completed the story of the life history of the ergot fungus.

Ergot occurs in most places where its hosts, numerous grains and grasses, are found, although it is rare in the southeastern United States. It is prevalent in every continent, throughout Europe, and in nearly all parts of the United States. It is particularly abundant in Siberia, the main source of the supply for the drug trade.

Importance. Ergot is primarily important on rye and certain pasture grasses because it lowers the yield, impairs the crop for seed purposes, and produces poisoning of man or animals eating infested crops or their products. Although as a rule yield decreases from ergot are relatively unimportant, losses as high as 20 per cent have been suffered in Russia. The annual losses for the United States in rye range between 50,000 and 350,000 bushels, mainly in the Lake States area. In the 1938 crop, for example, 382 carloads of rye were found to be ergoty, all but 22 of which were marketed at Minneapolis and Duluth. In grasses the yield may be affected to a serious extent, as in instances in Ireland and the United States where hay has been found to contain ergot to the extent of one-eighth of its weight.

Ergot poisoning of human beings is relatively rare today except in the

more backward sections of rye-eating countries. The loss to livestock is considerable but difficult to estimate. There are frequent reports of ergotism in small numbers of cattle, horses, and sheep, and occasionally serious local losses, largely due to feeding ergoty hay, as the feeding habits of animals would normally lead them to avoid ergot in the pasture.

In discussing the importance of the disease, mention should be made of the drug prepared from ergot which is an important constrictor of smooth muscle, indicated in cases of excessive bleeding and to aid childbirth.

Host Plants. There are about 20 species of *Claviceps* affecting various *Gramineae*, but as commonly understood, ergot refers to *Claviceps purpurea*. Of its cultivated hosts, rye is more commonly attacked than wheat, oats, and barley, although occasionally wheat, especially that designed for macaroni, may be appreciably injured. More than 100 species of grasses and sedges are hosts of ergot; the more important pasture grasses affected in the United States include the bluestems, wild ryes, Western wheatgrass, the bluegrasses, Indian grass, brome grass, twitch-grass, and barnyard grass. The fungus *Claviceps purpurea* consists of a number of physiologic races, specialized to attack particular hosts and not others.

Symptoms and Signs. The disease is recognized primarily by black, hard, banana- or horn-shaped sclerotia which protrude from the head in

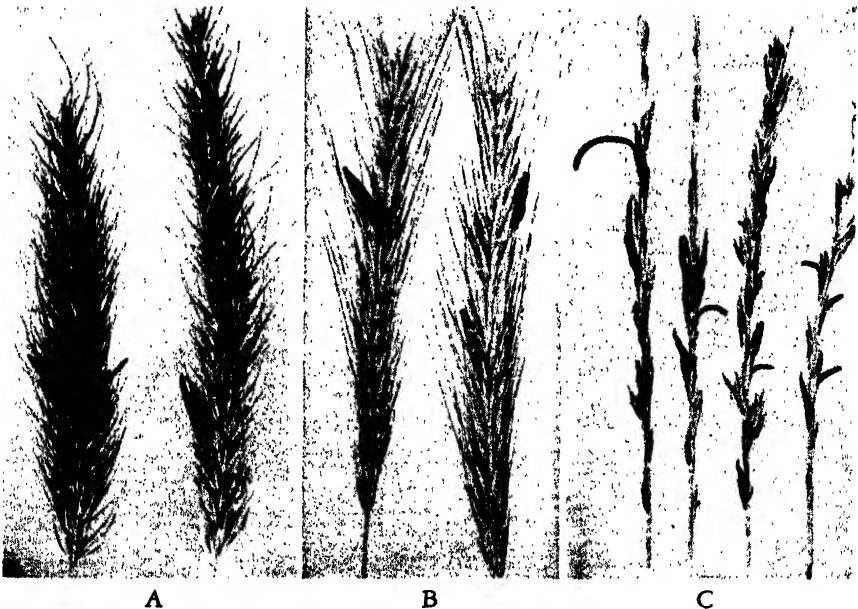


FIG. 52. Ergot. (B) In rye. (A and C) In pasture grasses. (A) Canada wild rye (*Elymus canadensis*). (C) Western wheat grass (*Agropyron Smithii*).



FIG. 53. Ergot-infested grass (*Andropogon ternarius*) with the saprophytic fungus *Cerebella* growing on the ergot honey dew. The presence of *Cerebella* is a useful indicator that the grass, which at this stage can be fed with safety, will later become poisonous when the ergot horns appear.

the place of normal grains (Fig. 52). They usually occur here and there in individual spikelets of the head. Earlier in the development of the head, before the sclerotia appear, a careful examination will reveal a sticky secretion oozing out from between the glumes, the "honey-dew." This is so inconspicuous in pasture grasses that it might be overlooked, except that frequently there is a saprophytic fungus, *Cerebella*, which feeds on the honey dew, forming black cushions over the infected parts, resembling a tiny black brain. These are a very useful indicator of ergot attack at a stage early enough to permit use of the infested grass as hay, before the dangerous sclerotia are formed. (Fig. 53.) In harvested rye grain the sclerotia are easily recognized as a contamination and important grading factor.

Etiology. The sclerotia of the ascomycete, *Claviceps purpurea*, are compact masses of fungus tissue that serve as resting or overwintering

bodies. In harvesting grain or in nature these fall to the ground where they overwinter, or they are introduced into a field by planting contaminated seed. In the spring they germinate, producing a globular, purplish stroma at the tip of a long, often twisted, stalk that may grow up through the soil for an inch or more. This stroma has the outward appearance of a golf ball, the irregularities representing mouths of many flasklike perithecia that are buried in the stroma (Fig. 54). The perithecia contain numbers of long asci, each in turn containing eight threadlike ascospores (Fig. 55). The ascospores are wind-borne to the host heads at the time of blossoming. On coming to rest on a stigma, the ascospore germinates, sending out an infection hypha which grows down the style and into the ovary much in the fashion of a pollen tube. The embryo is attacked and practically destroyed, being replaced by yellowish-white mycelium. With further development of the fungus there appears on the blossom the shiny, sweetish honey dew, which is cloudy from containing multitudes of conidia that have been produced by the mycelium (Fig. 55). The fluid attracts insects, which visit the heads and carry the conidia from diseased florets to healthy ones where the spores initiate new secondary infections and thus spread the disease. Later, the buried fungus mass grows larger, hard, and dark, and finally protrudes from the mature head as a sclerotium, the only form in which the fungus can overwinter. The sclerotium will germinate only after a period of cold, and can survive in storage two years or longer before germinating.

Epiphytology. Ergot infestation varies considerably from one year or locality to another, being very dependent on environmental conditions. Germination of the sclerotia and infection by the spores are believed to be favored by seasons with abundant spring moisture and by damp, low locations. Since infection takes place at blooming time, any factors, such as decreased sunshine, that lengthen the period in which the flowers are open, will have a tendency to increase the amount of infection. Irregular depth of seeding, such as results from broadcast planting and the use of



FIG. 54. Germinated ergot sclerotium, enlarged. The golf-ball-like stromata are mature, ready to eject the ascospores that initiate spring infections.

mixed grains for seed, has a similar effect in lengthening the period during which open flowers are present in the field.

Ergotism. Ergot poisoning or ergotism is caused by the powerful alkaloid, ergotoxine or ergotinine, which is contained with other poisons in the ergot sclerotia. This drug in small amounts produces a weakening of livestock without further symptoms. When larger amounts are consumed, gangrenous ergotism results, in which the blood supply to the extremities is insufficient. The consequence is a sloughing off of horns, hooves, teeth, ears, and hair, and ultimately a painless dry decay of the feet. The animal may become emaciated and die from the direct effects of ergot poisoning, or on the range it may more commonly die of thirst or starvation, being unable to move about. Other symptoms sometimes occur, such as blindness, convulsions, paralysis, and internal disorders. Poultry also may be affected.

Control. It is important that rye and grass seed be free of ergot sclerotia as the disease is usually introduced in a planted crop through contaminated seed. If ergoty seed must be used, the ergot sclerotia can be partially removed from the seed by fanning or screening, and completely by covering the seed with a 20 per cent solution of common salt (40 lbs. of salt in 25 gal. of water) or a 32 per cent solution of potassium chloride.

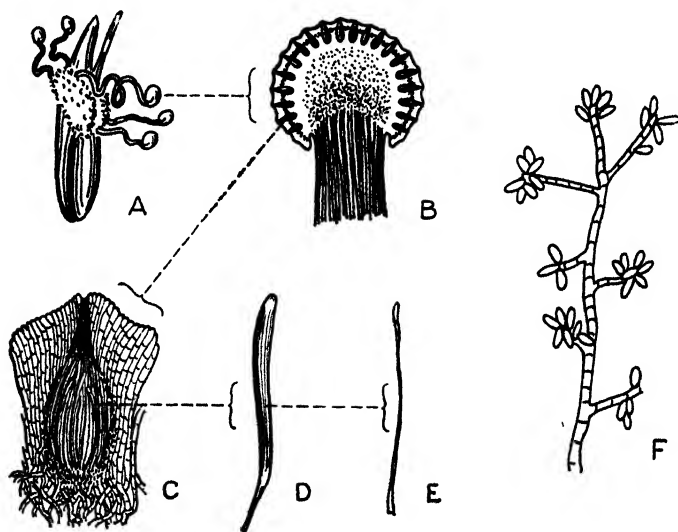


FIG. 55. Reproductive structures of the ergot fungus. (A) Germinating sclerotium with 5 perithecial stomata. (B) Single stroma, enlarged section showing many perithecia. (C) Single perithecium containing many asci. (D) Single ascus with 8 ascospores. (E) Single ascospore such as initiate primary infections. (F) Conidia which are produced with honey dew in grain or grass blossoms and serve for secondary spread of the fungus. (B-F after Tulasne and Gäumann.)

In such a solution the seed sink and the sclerotia float to the top where they may be skimmed or poured off. The seed are then washed in water and dried. Rye should not be planted until the third year in a field in which the preceding crop was ergoty, and efforts should be made to destroy ergoty grasses in furrows and fence rows.

In native pastures ergot can be controlled by cultural or cropping practices, such as burning over the patches of affected grass, by harvesting the grass for hay before the sclerotia form, or by close grazing to accomplish the same purpose. Grazing livestock usually will avoid ergotized heads unless these are softened by moisture and ergot-free grass is scarce, as when the pasture or range is covered by a light, melting snow. Under such conditions, or wherever there is heavy ergot infestation, livestock should be kept away from infested areas. The feeding of ergoty hay is even more dangerous, since in this case the animal has no choice and cannot avoid eating the sclerotia. In cases of heavy infestation of rye the grower might consider the possibility of harvesting the sclerotia for the drug which often brings a high price. A way has recently been found of deliberately infesting rye with ergot. Grass ergot could not be used for this purpose, even though it is rich in the needed drug, because the U. S. Pharmacopoeia limits ergot, by definition, to the fungus from rye.

Powdery Mildew of Cereals (*Erysiphe graminis*)

History and Distribution. The powdery mildew of cereals and grasses has been known as a disease since early historical times and as a subject for investigation since 1815. It occurs apparently wherever cereals are grown, particularly in the north and south temperate zones. In the United States it has attracted attention especially in fall-sown barley in the southern and eastern Mississippi Valley States and on the Pacific coast.

Importance. Because of its common occurrence and the infrequency of its epiphytotic development, powdery mildew often is regarded as a benign and harmless disease. Yet, according to Melhus and Kent, "it may produce a great deal of damage when favorable environmental conditions allow widespread infections early in the season." Whetzel also held that its annual widespread occurrence suggests that it is probably far more important as a factor in reducing the yields of wheat, oats, barley, and rye than plant pathologists or growers generally appreciate. The injury takes the form of yellowing and premature killing of the lower leaves and excessive respiration which in turn retards the development of the plant and interferes with the filling of the grain, or in severe cases even prevents heading.

Host Plants. More than 50 species of *Gramineae* are attacked by

Erysiphe graminis, including barley, wheat, oats, rye, and many wild and cultivated grasses. The pathogen exhibits physiologic specialization, with seven varieties each of which is adapted to certain hosts. The principal



FIG. 56. Powdery mildew of wheat.

varieties on barley, wheat, oats, and rye are *E. graminis* varieties *hordei*, *tritici*, *avenae*, and *secalis* respectively. Not all varieties of the cultivated grains are equally susceptible. Grasses vary from highly susceptible to highly resistant, and within grass species there may be great variation in mildew reaction of individual plants, making possible the selection of resistant grass strains. The more resistant cereal varieties recorded include:

Barley: Arlington Awnless, Bolivia, Chilean, Consul, Duplex, Kwan, Sunrise, and Wong.

Winter wheat: Chiefkan, Fulcaster, Fultz, Fultz-Mediterranean, Hardired, Hope-Hussar, Hungarian, Hussar, Hussar-Hohenheimer, Illinois No. 2, Kharkov, Mediterranean, Mediterranean-Hope, Michigan Amber selections, Penquite, Redhart, Relief-Ridit, Turkey selection, and Wisconsin Pedigree No. 2.

Spring wheat: Merit, Progress, and Renown.

Rye: Abruzzes selection (also stem rust and leaf rust resistant).

Symptoms and Signs. Powdery mildew is recognized by white, mealy or fluffy, somewhat circular mycelial coatings, largely on the leaves, sometimes so numerous as practically to cover the leaf (Fig. 56). Later the mycelium becomes brownish and is sometimes studded with tiny brown fruiting bodies, the perithecia. The mealy appearance is due to myriads of conidia over the surface of the mycelium. The fungus does not penetrate deeply, hence the tissue under the lesions is green until advanced stages when the entire leaf becomes yellow and

may die. The plants are likely to be stunted, with light grain or even blasting or lack of heading.

Etiology. The fungus, *Erysiphe graminis*, an ascomycete, is an obligate parasite. The perithecia, formed late in the season, are one means of overwintering, although where the hosts are in the vegetative stage throughout the winter, the fungus may pass the winter in the parasitic, conidial stage or as overwintering mycelium in leaves. The perithecia overwinter in decaying host tissues. In the spring ascospores develop in 8 to 10 asci within each perithecium. There is no opening to the perithecium, and this particular form of perithecium is often termed a cleistothecium (see Fig. 36). Under pressure of the developing asci it bursts, liberating the ascospores by forcible discharge. These may be wind-borne to new host leaves and there infect. The ascospore germinates forming an infection hypha which, coming into contact with the epidermis, flattens out to form a sucker-like disk (appressorium) attaching the hypha to the epidermis. From the center of the appressorium a fine peg forces its way through the cuticle, then expands to form fingerlike feeding organs (haustoria) within the epidermal cells. By progressive growth and branching, more and more epidermal cells are involved but the fungus does not penetrate deeper into the leaf tissues. After a period of feeding, and development of surface mycelium, erect hyphae (conidiophores) are extended upward from the mycelium, each bearing a chain of thin-walled, barrel-shaped conidia. As the terminal conidia mature and blow away, new ones are formed at the base of the chain. The conidia, produced in great numbers, initiate the secondary cycles and thus spread the disease. With the advance of the season, sexual union occurs between specialized cells of the mycelium and as a consequence the globose, hard, thick-walled, dark perithecia appear. The perithecia are ornamented with characteristically formed appendages, and the various genera of powdery mildews are separated by the character of the appendages and the presence of one or of more than one ascus per perithecium.

The injury to the host tissues is due to exclusion of light, excessive loss of water from infected leaves, and excessive respiration, which may reach 650 per cent that of normal plants, burning up the carbohydrates that should be used for growth and seed formation.

Epiphytology. While the conidia of *Erysiphe graminis* do not require a film of water for germination, and can even germinate at zero humidity, they germinate best in a moist atmosphere (above 95 per cent). This is the main factor limiting the development of the disease in dry locations. As with the rusts, this obligate parasite is most destructive on vigorous and succulent plants. Temperature is a factor principally as it increases or

decreases the vigor of the host, rather than through its direct effects on the parasite. Spore germination, germ tube growth, and infection are greatest at 68°F. Some infection occurs between 30° and 77°F., but the fungus cannot tolerate temperatures above 85°F. Alternating temperatures induce perithecium formation, and alternating wetting and drying favors development of the ascospores. Abundant nitrogen fertilization favors powdery mildew; potassium and phosphorus decrease it. Light is essential to infection, evidently because the fungus can infect only those tissues in which photosynthesis is active. Summarizing, the disease is most severe under moist conditions and those favoring a vigorous, luxuriant growth of the host. As these conditions are not realized regularly from year to year and from one location to another, the disease tends to appear in destructive epiphytotics on some occasions, while at other times it is of inconsequential occurrence.

Control. As indicated above, there are numerous varieties of the small grains that exhibit resistance to all or most of the strains of powdery mildew. In areas of infestation, these resistant varieties, or the newer varieties constantly being produced from mildew-resistant breeding stocks, offer the best means of control. In grain-breeding programs, powdery mildew resistance is being considered, along with other characters, for incorporation into newer synthetic varieties.

Powdery mildew may be effectively controlled on susceptible cereals by applications of sulfur dust. In normal field culture, this would ordinarily not be practicable, but dusting has its use in protecting valuable seed plots or show grain. Sulfur dust is effective also against other small grain diseases, such as the rusts, and since the rusts and powdery mildew develop under similar conditions, the combination of rust and mildew might at times warrant airplane applications of sulfur, now available at reasonable cost. If leaf-chewing insects are a factor, an arsenical could be economically applied with the sulfur.

Powdery Mildew of Roses (*Sphaerotheca humuli* and *S. pannosa*)

On some of the ornamentals, powdery mildews may be extremely destructive diseases because of their virulence, and because even light mildew attacks may ruin the ornamental value of the hosts. This is particularly true of the powdery mildews of zinnias, phlox, and roses, for example. The former are two killing diseases, the latter causes serious disfigurement, devitalization, and inhibition of blossoming. All aerial parts of the rose are affected, especially new growth and bud clusters. The familiar mealy, white mycelium covers affected parts, and leaves are often much wrinkled, somewhat discolored, distorted, and reduced in size (Fig.

57). Some of the climbing roses with large clusters of small blossoms, such as Dorothy Perkins, Crimson Rambler, and Excelsa, are often so badly mildewed that the buds are ruined and the plants seriously impaired as ornamentals. The fungus overwinters either by perithecia on fallen leaves, or as conidia, and constant successions of conidia under moist conditions in the spring produce the heavy infestations so commonly seen. Highly nitrogenous soil, such as often accompanies rose culture, promotes the succulent, vigorous type of growth most favorable to the disease.

Satisfactory control is obtained by periodic use of a fungicide. In the past, sulfur dust, sometimes colored green to make it inconspicuous, has been commonly used, but this does not always give control, and in hot seasons it may be somewhat injurious. Sprays of wettable sulfur are somewhat more effective. Recently Fermate has given excellent results against mildew used at the rate of 1 lb. in 100 gal. of water with a good spreader-sticker. In extensive research in Texas, the best rose fungicide appears to be dusting sulfur plus insoluble copper dust, 1:9, with little choice between the coppers. Other rose growers prefer copper oxide (Cuprocide 54) as a spray. In greenhouses the disease is controlled sometimes by daubing sulfur paste on the steam pipes, where it volatilizes, but there is danger to the foliage if the pipes become too hot. Roses vary in mildew susceptibility, but desirable varieties need not be rejected in view of the success of chemical control. Progress is being made in breeding mildew-resistant climbing roses.

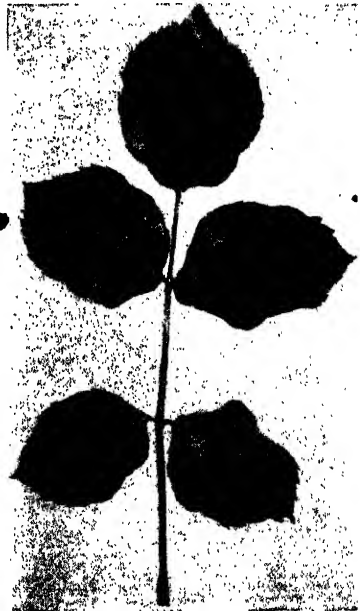


FIG. 57. Powdery mildew of roses. Powdery mildews of other hosts are very similar in appearance. (Courtesy, P. P. Pirone, N. J. Agr. Exp. Sta.)

Other Powdery Mildews

The powdery mildew of legumes, *Erysiphe polygoni*, is best known on clovers, peas, beans, alfalfa, and cowpeas, although it attacks some 200 species in 90 genera of plants. It seems to have been introduced about 25 years ago, and in 1922 it caused such serious damage to clover in the eastern United States that the epiphytotic was regarded as the most note-

worthy plant disease outbreak of the year. Since that time it has continued to be quite injurious. As yet there is no generally satisfactory control for powdery mildew in clovers and other field legumes, although sulfur dusting, where the expense is warranted, will check the disease. Some European clovers are resistant and are the basis of present-day breeding for mildew resistance. Sulfur dusting is practical and often essential for the protection of peas.

The powdery mildew of cucurbits, *Erysiphe cichoracearum*, also came into prominence about 20 years ago, when it broke out in the Imperial

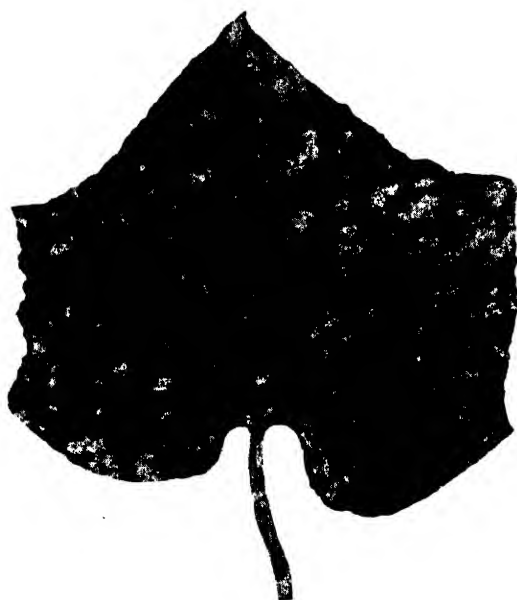


FIG. 58. Powdery mildew of cucumber, a common problem in cucumbers under glass.

Valley of California, the leading melon district of the country, reducing the crop by 5000 carloads in 1926. Within 10 years it looked as though the problem had been solved by the development of Powdery Mildew Resistant Cantaloupe No. 45, from a cross between the susceptible commercial variety, Hale's Best with a resistant variety from India, followed by backcrossing with Hale's Best and repeated selection. No. 45 met all requirements of growers, shippers, and consumers, and showed striking resistance to mildew. But the mildew fungus did not give in. In 1938 there appeared a new physiologic race of mildew which could attack No. 45, and again the growers turned to the breeders for help. Melons that were tolerant to both old and new mildew races were made available to growers, but the breeding must continue. The disease can be controlled by sulfur

or preferably insoluble copper dusts or by fungicidal spraying. (See Figs. 58 and 211.)

The powdery mildew of apple, *Podosphaera leucotricha*, occurs widely but is most destructive in the irrigated regions of the Pacific Northwest. The same fungus attacks a number of other relatives of the rose family. Mildew is easily recognized on apple twigs and leaves as a white, powdery coating of conidiophores and conidia. The leaves are stunted or killed and this devitalizes the trees, lowering production or even causing crop failure the following season. The fruits may be stunted or russeted, lowering their grade. The fungus overwinters as dormant mycelium on twigs or in buds, and the ascospores, formed as in the cereal mildew, seem to play little part in the life cycle. Control depends on removing the mildewed twigs at the time of winter pruning and spraying. The spray program outlined for apple scab (p. 102) gives sufficient protection against mildew.

The powdery mildew of gooseberries, *Sphaerotheca mors-uvae*, is the most important disease of this crop and at times is a factor limiting production. It is very destructive in Europe where the disease was introduced from America. Currants also are attacked. The disease is recognized by the familiar white, powdery coating of leaves, green shoots, and especially the berries, which are conspicuously stunted, dark discolored, and unmarketable. For control it is necessary to use three applications of lime sulfur, 2-100, Bordeaux mixture, 3-5-50, wettable sulfur, or sulfur dust.

Brown Rot of Stone Fruits (*Monilinia fructicola*)

History and Distribution. Brown rot has been known as the most generally destructive disease of peach, plum, cherry, and related stone fruits in America. It occurs wherever these fruits are grown in the United States and Canada, and is a problem also in Australia, Tasmania, New Zealand, and possibly South America. A very similar disease, often confused with this one but caused by a distinct species of *Monilinia*, *M. laxa*, is common in Europe, in the Pacific States, and in British Columbia, and has been found in Wisconsin but not elsewhere in the central and eastern United States. Brown rot has been troublesome in the United States for at least 150 years. It was first studied scientifically in 1881 by the mycologist Peck. The sexual overwintering stage was determined in 1883. Control by the use of self-boiled lime sulfur was discovered in 1908-1910, and recent studies have been largely concerned with the etiology of the disease and its control by improved spraying practices, sanitation, and the use of varietal resistance.

Importance. Brown rot is most destructive in the more humid areas although it is not limited to these. The average annual loss in the peach

crop exceeds \$5,000,000 and the loss in Georgia alone has reached \$2,000,000, or 40 per cent of the crop, in some years. At times the losses for individual states are nearly 100 per cent, as in Alabama in 1897, while state losses of one-third to two-thirds of the crop have not been uncommon. In addition to losses in the orchard, many carloads of stone fruits in transit show considerable brown rot, sometimes reaching 90 per cent, and similar destruction is encountered in markets and homes.

The losses from brown rot are of several types. The disease affects the blossoms, causing barrenness, from which point it may work down into the twigs, killing them, and it occasionally causes large leaf spot lesions. It may affect the green fruits, but the principal damage is on the ripe fruits, which are very quickly rotted, either in the orchard or in storage or transit.

Host Plants. The most important hosts of the brown rot fungus are the stone fruits—peaches, plums, cherries, apricots, almonds, and nectarines. Many wild species of *Prunus* are affected and the disease is sometimes found, although not usually serious, on other plants in the *Rosaceae* such as quince, apple, and pear.

Considerable variability in attack has been observed among commercial varieties of stone fruits. The reason why a number of the newer varieties have replaced older ones is their lower damage from brown rot as observed by growers. In general it may be said that the early and light-colored peaches, the Japanese, light-colored, and thin-skinned plums, and the sweet and white cherries are most severely attacked. The greater freedom from attack of other varieties is probably not due so much to inherent resistance as to their growth habits that permit them to escape rather than resist the disease, e.g., by maturing when weather conditions are unfavorable for brown rot infection.

Symptoms and Signs. *In blossoms*, the disease causes cessation of growth, reduction of size, wilting, pallor of the calyx, and killing of the petals, all of this inhibiting fruit development.

In twigs, the fungus grows from the blossom or fruit pedicel into young twigs, killing them back much as in fire-blight of apple and pear.

In leaves, the entire leaf may wither as a result of twig infection, or it may rarely show large, circular, dead spots that drop out.

In limbs, cankers may result as the fungus passes down the twigs into the larger wood. The bark is killed, cracks open, and exudes gum. This may be followed by callusing, only to enlarge the following years, so that ultimately large limbs may be girdled and killed.

In fruit we have the most familiar symptoms (Fig. 59). Fruits can be attacked at any time after they are half grown but usually infections are noticed after ripening. Lesions first appear as small, round, well defined,

light brown spots which enlarge so rapidly that within a few days the entire fruit is rotted. Beneath the lesion the decayed flesh is soft and dark brown. The surface of the lesion is covered with fruiting tufts (sporodochia) of



FIG. 59. Brown rot of peach, showing masses of spores (conidia). (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)



FIG. 60. Brown rot of peach. Production of apothecia from overwintered, buried mummy.

the fungus, consisting of numerous conidiophores bearing chains of conidia (*Monilia* stage). Often these tufts are arranged in concentric rings, marking diurnal fluctuations in temperature. On plums the lesions are sometimes difficult to detect in early stages because of the dark colored

and thick skin. After fully decaying, the fruit shrivels and becomes a dry, hard, wrinkled mummy, hanging on the tree or falling to the ground.

Etiology. The ascomycete, *Monilinia fructicola*, overwinters in mummies as sclerotial masses, and to a lesser extent in limb or twig cankers. The primary infections in the spring, which usually occur on the blossoms, are due either to conidia from the cankers or mummies hanging on the trees, or to ascospores that are formed in cup-shaped apothecia. The latter, borne on stalks, are produced by the germination of the sclerotia in the mummies partly buried in the soil (Figs. 60, 61). Lining the inner surface of the apothecium are many asci which shoot out clouds of ascospores in visible puffs, which then may be wind-borne to infection courts—the stigmas of blossoms. Here the germ tube of the germinating spore, making use of the nectar in the blossom as foodstuff, penetrates and infects the floral parts. Soon conidia are formed over these from the feeding mycelium, and these begin a series of secondary cycles. Meanwhile the mycelium advances into the twigs, producing twig blight. As the fruits mature conidia are wind-borne to them, infecting usually through wounds, especially the punctures made by the curculio and oriental fruit moth; 90 per cent of fruit lesions develop about such wounds. As the fungus sporulates on the fruits countless new conidia are formed, available for secondary cycles in the orchard and in storage. In the act of decay the advancing mycelium gives off a toxin that kills the cells in advance, liberating food materials for its use. The fungus continues to

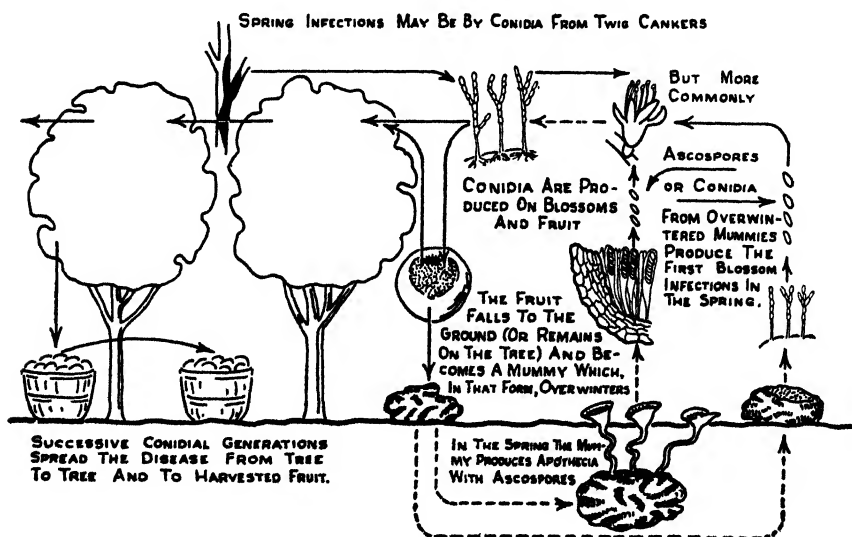


FIG. 61. The life cycle of brown rot of stone fruits. (Section of apothecium after Norton *et al.*)

produce conidia on the surface of the rotted fruit until it dries, shrinks, and becomes a mummy, in which the sexual processes preceding apothecia formation take place.

Epiphytology. *Monilinia fructicola* is active at temperatures from 32°F. to 90°F. with a pathogenic optimum at about 75°F. This temperature relation would rarely limit its activity. Moisture at infection periods is the most important single factor predisposing to epiphytotics, spring rains promoting apothecial development, conidial spread, and spore germination. The spores will germinate only in a film of water. Blossom blight requires high humidity. Low lying orchards suffer most because of slow drying after rains. Well-pruned, open trees suffer less for a corresponding reason. Wounds, caused by other diseases, hail, insects, and birds, play a large part in permitting infection. Apothecial production is most favored in acid soil. Frequently sclerotia do not produce ascospores until the second year after their formation. Altogether, the fungus is capable of developing under a wide range of environments, provided adequate moisture is available, and local or periodic epiphytotics are largely correlated with spring precipitation.

Control: SPRAYING OR DUSTING. The most important part of the control program for brown rot is spraying or dusting.

Applications. Various spray or dust materials and programs are preferred in different states and for the different stone fruits. The following are representative practices.

a. Dormant spray. A strong dormant spray of lime sulfur (12-100) or Bordeaux mixture (6-6-100) is sometimes considered helpful in control of twig and blossom phases of brown rot, but the twig cankers are quite resistant to fungicides and in a number of states this spray is not regarded as very effective against brown rot although it is useful against peach leaf curl, scale, and mites. In California, against *Monilinia laxa* which is particularly important in its attack on twigs and blossoms, good control is obtained with a delayed dormant spray of copper-lime-monocalcium arsenite-oil, followed by an early bloom spray of 3-4-50 Bordeaux.

b. Prebloom or pink bud spray. At this stage a spray of lime sulfur, 1-50, has long been recommended. Wettable sulfur or flotation sulfur may be substituted.

c. Other early sprays. In some states, as protection against the early stages of brown rot one or more of the following applications is recommended: bloom spray of wetttable sulfur; petal fall spray of zinc sulfate, 3 lbs., spray lime, 6 lbs., lead arsenate, 2 lbs., and, if brown rot was severe the preceding year, wetttable sulfur, 6 lbs., all in 100 gal. of water; shuck spray of the same materials as the petal fall spray.

d. Cover sprays and dusts. These are of most importance in protecting the fruit. Usually three or more applications at 10-day intervals before harvest are recommended, using wettable sulfur or fixed copper sprays, or sulfur dust with or without lime. Lead arsenate may be used in the earlier cover sprays, where it is important in reducing insect wounds which permit brown rot entry, but it cannot be used in the later cover applications because of the poisonous residue on the fruit. On cherries, lime sulfur, 1-50, is used sometimes for these applications.

The new organic fungicides such as Phygon and Zerlate are proving very useful in the preharvest sprays, often excelling the older standard materials. Tetramethylthiuram disulfide (Tersan, etc.) and Dithane with zinc sulfate and lime also have given good brown rot protection.

Stone fruits are particularly liable to spray injury if the recommended spray schedules are not followed closely. Excessive applications must be avoided, and the spray should be applied as a fine mist, not a drench. The spray injury hazard and the most effective spray programs vary from one area to another, and orchardists should constantly make use of the latest recommendations of local experiment stations and agricultural advisers.

SUPPLEMENTARY MEASURES. While spraying is most important in brown rot control, there is value also in sanitary measures, to remove sources of inoculum. Though it may be impractical to attempt to remove the smaller twig infections, the larger cankers can be cut out and burned or surgically treated, and valueless infected trees near the orchard can be removed. More important is the removal of mummies. These can be knocked from the tree at picking time. Brown-rotted fruit should be culled out and destroyed, and the mummies on the orchard floor removed by raking or disking under, or eaten by hogs turned into the orchard for the purpose. A disking in the spring just before the blossoms open is helpful in disturbing the mummies and thus markedly interfering with their ability to produce apothecia. Brown rot storage and transit decay is minimized by selecting only sound fruit for the purpose, avoiding bruising in picking and packing, and maintaining the storage temperature as near to 32°F. as possible.

Cherry Leaf Spot (*Higginsia hiemalis*)

The most frequent and destructive foliage disease of all common varieties of cherry is the leaf spot, sometimes known as shot-hole leaf spot or yellows. The spots are circular, first purplish, later brown, then falling out to give the shot-hole effect (Fig. 62). Where the spots are numerous the leaves commonly turn yellow and fall by midsummer or

earlier. Loss of leaves after harvest may seem unimportant to the grower, but has great effect in reducing the crop of the following year. The fungus, *Higginsia hiemalis*, overwinters as a saprophyte in fallen leaves. In the spring it produces needlelike ascospores in sessile, disk-shaped apothecia. In moist weather, these produce the primary leaf infections, followed by the production of masses of summer spores (conidia) on the lesions, serving for secondary spread. Control is aided by disking under the fallen leaves before blooming, but the main control measure is the spray schedule. The spray schedules for cherry leaf spot vary greatly from one state to another as the following examples show, and again emphasize the importance of securing and following local recommendations. In general, four applications are used, petal fall, shuck, first cover, and post harvest. Uniform tests in Virginia, Pennsylvania, and West Virginia (incidentally an excellent example of research teamwork) have indicated early season applications of lime sulfur and later ones of Bordeaux mixture as most effective and least injurious in that area. Fixed copper may be used in place of Bordeaux in the latter sprays. In Wisconsin where the leaf spot problem is serious, where dilute Bordeaux is ineffective, and where strong Bordeaux is effective but injurious, a good schedule has been found to be four applications of Bordeaux of different strengths, 6-8-100, 3-4-100, 1½-2-100, and 6-8-100, respectively.

Frequently lime sulfur, 2-100, has been satisfactory for all four sprays, or, for postharvest spray, the substitution of wettable sulfur, 6-100, lime sulfur, 1 gal. plus 3 lbs. of fused sulfur, per 100 gal., or Fermate 1½ lbs. plus lime and spreader.

There are conflicting reports on the value of the organic sprays for cherry leaf spot control, excellent results in some areas and poor ones in others having been obtained with Fermate, Dithane, Isothan Q15, and Methasan (Zerlate). Phygon has performed well in Virginia and Pennsyl-

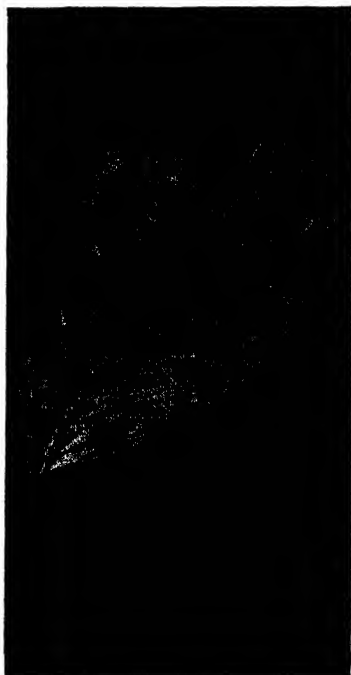


FIG. 62. Cherry leaf spot showing various stages leading up to shot holing. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

vania. Undoubtedly some of these will have an important place in future leaf spot control in some areas, but local recommendations for their use should be followed.

Some of the understocks used in cherry propagation are very susceptible to leaf spot, and spraying in the nursery can be avoided by using piece-root grafts on these instead of budding.

Alfalfa Leaf Spot (*Pseudopeziza medicaginis*)

One of the common leaf spot diseases of alfalfa is due to the ascomycete *Pseudopeziza medicaginis*. This can be distinguished from other alfalfa leaf diseases by the fact that the spots, although they may be numerous, are quite small (2 to 3 mm.), circular with irregular margins, black, and show, usually on the upper leaf surface in the center of the spot, a tiny round, raised disk, the apothecium. When young seedlings are attacked, they may be defoliated or entirely destroyed. In older plants the leaves, especially the lower ones, are shed in the field or in harvesting, until the hay may consist largely of naked stems.

The only spores in the life cycle of *Pseudopeziza medicaginis* are the ascospores, produced in the apothecia on the leaf spots. These serve both for primary infections and for secondary spread during the growing season. The fungus overwinters either as mycelium or as apothecia in the dead leaves. Infection is said to be relatively independent of weather but favored by cultural conditions that produce early shading of the ground. Losses can be prevented by harvesting frequently, before leaf-shedding has become injurious. Seed selection and seed treatment are of no value as the disease is not seed-borne, and no resistant varieties of alfalfa, the only host, are known.

Anthrachnose of Cane Fruits (*Elsinoë veneta*)

Anthrachnose or "gray-bark" is the commonest disease of raspberries. It is most destructive on black and purple varieties, and is sometimes damaging to blackberries, red raspberries, and other cane fruits. Canes of affected plants late in the season are light gray from the presence of numerous, small gray lesions, dotted over with black acervuli, or clusters of conidiophores and conidia (Fig. 63). In severe attacks the canes may crack open. The young lateral growth is attacked and often killed. On the leaves, the fungus causes tiny yellow lesions with raised brownish margins. When these are numerous the leaves may fall. Lesions similar to those on the canes also are found on petioles, peduncles, and pedicels, and rarely the fruits are destroyed, becoming brown, dry, and woody. Continued attack weakens the plants, makes them more susceptible to

winter injury, reduces production, and may even kill the plants. In the autumn, primitive fruiting bodies are formed that mature ascospores, usually in the spring. These initiate the primary infections, while secondary spread occurs by means of the conidia during the earlier part of the growing season. Infections are favored by damp weather in the early part of the season, and losses are greatest when this is followed by unfavorable growing conditions the second year.

Control is achieved through certain cultural practices and spraying. When cane fruits are planted stubs of old canes ("handles") should be cut off below the ground level. In the spring pruning heavily infected canes should be removed, and all fruiting canes after harvest. So far as possible select healthy planting stock, avoid damp locations as planting sites, keep weeds under control, as these make moist air pockets which favor infection, and maintain soil fertility to stimulate new healthy cane growth. Some black raspberry varieties have anthracnose resistance, for instance, Naples and Quillen, and hybrids of the latter with Black Pearl, developed in Iowa.

Spraying for anthracnose control involves two or more applications, a dormant or delayed dormant spray of 1-10 to 1-20 liquid lime sulfur, applied when the buds are beginning to break, and one to three cover sprays of 3-3-50 Bordeaux mixture or lime sulfur 1-50, covering the canes thoroughly. Fermate in the growing season and Elgetol as an eradicant spray have given good results in anthracnose control in New York.



FIG. 63. Anthracnose lesions on shoots of black raspberry. (Courtesy, R. F. Suit, N. Y. Agr. Exp. Sta.)

Black Spot of Roses (*Diplocarpon rosae*)

The large, disfiguring lesions of black spot are familiar to every grower of roses, as the disease is almost universally present in rose culture, outdoors and in the greenhouse (Fig. 64). The spots are up to $\frac{1}{4}$ inch or more in diameter, fringed in outline, and sprinkled with very tiny, inconspicuous fruiting bodies. In severe cases the leaves become yellow and

fall, and even light attacks seriously impair the beauty and value of the plants. The fungus, *Diplocarpon rosae*, is an ascomycete, with nonsexual reproduction by means of conidia formed in clusters (acervuli) on the new lesions, and with an ascospore stage developing on the fallen leaves in the spring after the fungus has hibernated in the leaves as a saprophytic mycelium. The disease develops most destructively under cool conditions (60°F. to 70°F.) when abundant moisture is available. The spores are not easily detached by wind and are spread mainly by rain, splashing water, or greenhouse syringing.

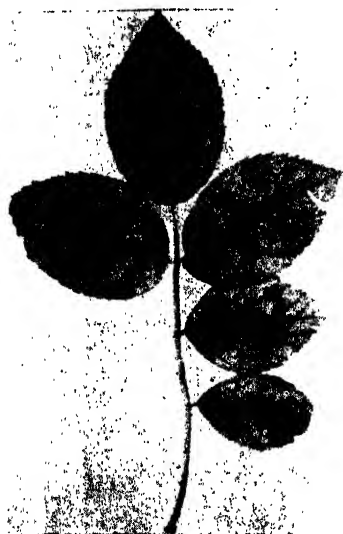


FIG. 64. Black spot of roses. Two leaflets have fallen and the others are so disfigured that such plants would be worthless as ornamentals. (Courtesy, P. P. Pirone, N. J. Agr. Exp. Sta.)

The disease can be controlled by fungicidal spraying or dusting at 1- to 2-week intervals after the disease appears. Sulfur dust has been used extensively in the past but now is largely replaced by Fermate, 10%, plus sulfur, 90%, used as a dust, copper oxide (Cuprocide), or copper-sulfur dust, 1-9. Phygon also has promise as a dust mixed with talc. In the greenhouse Fermate gives good control, but fungicides may not be needed if the house is kept dry and syringing is avoided. Syringing is sometimes used in red spider control, but a more effective way to keep down red spiders is by hexaethyl tetraphosphate fog.

Numerous rose varieties are resistant to black spot, and their use avoids spraying or dusting. Lists of these are to be found in the *Plant Disease Reporter*, **24**, 478 (1944), the *American Rose Magazine*, **5**, 144 (1944), and the *Iowa Agricultural Experiment Station, Annual Report for 1943*.

Peach Leaf Curl (*Taphrina deformans*)

History and Distribution. Leaf curl is known as a serious disease wherever peaches are grown. In the United States it was introduced prior to 1852, and now occurs in all important peach districts.

Importance. Leaf curl causes several types of losses. It destroys the new leaves in the spring, which necessitates a second foliation, draining the vigor of the tree. This renders the tree more susceptible to winter injury and reduces the fruit set the following year. Loss of leaves for

several successive years will kill the tree outright. The young fruits are attacked and fall prematurely. These injuries lead to losses estimated at 2.5 to 3 million dollars annually in the United States crop. With the general adoption of spraying for leaf curl control, the damage has been substantially reduced, but the cost of spraying must be included in the loss caused by this disease.

Host Plants. *Taphrina deformans* attacks only the peach and its derivatives, such as the nectarine and peach almond. A similar fungus causes



FIG. 65. Peach leaf curl.

plum pockets, a disease much like leaf curl in etiology and control. Peaches differ in susceptibility to leaf curl, but varieties resistant in one place may be susceptible in another. Some of the best commercial varieties are highly susceptible, and in view of the ease and efficiency of control by spraying, no extensive use is made of varietal resistance to leaf curl in peach culture.

Symptoms and Signs. The young leaves are arched and reddened as they emerge from the bud. Soon they appear very much curled, twisted, or puckered, thick and brittle (Fig. 65). Affected portions are first pale yellow or whitish, later covered with a silvery bloom. A few of the affected leaves remain on the tree, but many of them die and fall, and the symp-

toms become less apparent as the season progresses, the new, second crop of leaves being unaffected. New twigs may be swollen, pale, or even killed back. Young fruits are distorted, scabby, cracked, and soon fall. Mature fruits are occasionally affected, showing striking reddish discoloration and atrophy. In plum pockets, a very similar disease, the fruits are transformed into large, hollow, irregular bladders, and the twig tips, terminal buds, and leaves are grossly distorted.

Etiology. *Taphrina deformans* differs from all of the preceding ascomycetes in that the asci are not found in a fruiting body but are in a naked layer over the surface of affected parts. There is no conidial stage in the ordinary sense. Soon after release from the ascus the ascospores germinate to form a yeastlike colony which lives saprophytically on the bark of the

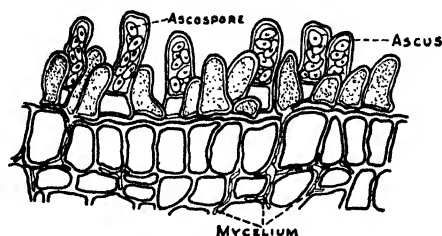


FIG. 66. Peach leaf curl. Asci and ascospores on the leaf surface and knobby, jointed mycelium between the cells. (After Pierce and Atkinson.)

tree and is the source of infection the following spring. At that time, when the buds are swelling, the yeastlike spores infect the young bud tissues, forming a peculiar, knobby intercellular feeding mycelium that gives off a toxin stimulating the leaf to distortion. Eventually a layer of asci appears on the leaf surface (Fig. 66).

Epiphytology. Leaf curl is favored by cool weather, 50° to 68°F., which at the same time slows down the development of the young shoots, giving the fungus a longer period of activity. The spores are quite resistant to drying and summer temperatures, although a very hot summer reduces infection the following spring. High humidity in early spring favors the disease both by permitting the spores to germinate and by retarding the development of the young leaves. Any other factor retarding spring foliation, such as wet soil, also increases leaf curl damage. Sudden appearance of the disease in orchards where it was previously unknown may be due to new parasitic activity on the part of the fungus which previously had existed in the orchard only in the saprophytic, yeastlike stage.

Control. Leaf curl and plum pockets are controlled easily by spraying at any time in the dormant season. Usually lime sulfur is recommended, the 32°-Baumé concentrate being diluted 1-20 or, if San José scale is a factor, 1-8. In absence of scale, Bordeaux mixture 5-5-50 may be used. In Washington and New York tests, Fermate and Elgetol have both given control equal to or better than standard sprays. The application can be made at any time in the dormant season after leaf fall, but freezing weather should be avoided. Delaying the spraying until spring is dangerous, since the ground in the orchard is often too soft to permit timely spraying in the spring. Contrary to statements often encountered, good control may also be obtained by using a weaker spray, such as lime sulfur 1-50, Bordeaux 2-4-100, or wettable sulfur, 16 lbs. per 100 gal., applied after the buds have swollen, until the leaves are protruding as much as one inch from the buds. It is important to coat every part of the tree evenly and thoroughly with a fine spray.

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Chapter 7

Diseases Caused by Imperfect Fungi

The classification of fungi is based on their mode of sexual reproduction. There is a large group of fungi in which the sexual stage may be lacking or at least not yet discovered. These are called "Imperfect fungi" (*Fungi imperfecti*). It is an artificial grouping, a catch-all containing nonsexual stages of ascomycetes, basidiomycetes, and phycomycetes. As the perfect stages of imperfect fungi are discovered they are transferred to their proper genera in the ascomycetes, basidiomycetes, or phycomycetes. This is one of the reasons we find duplication of technical names in fungi, where the name of the imperfect stage was first given, and later the entirely distinct name of the sexual stage after its discovery. If the name of the imperfect stage is the one found commonly, the imperfect name is often commonly used, just as Mrs. Tom Brown is still known to her old friends by her maiden name of Mary Smith.

The imperfect fungi are classified first according to the presence and type of nonsexual fruiting body, and second according to the type of spore produced. We find among the imperfect fungi some which have no spores at all (*Mycelia sterilia*), and others in which the conidia are borne at random over the surface of the mycelium (*Hyphomycetes* or *Moniliales*), in masses or acervuli (*Melanconiales*), or in flasklike pycnidia (*Sphaeropsidales*). Representative fruiting structures are shown in Fig. 37. Although many of the imperfect fungi are saprophytes, each of the classes contains destructive pathogens, in fact the imperfect fungi rank as a major group in producing plant diseases. Roughly subdividing the imperfect fungi according to life history and methods of control, we will consider first those that are primarily soil-borne, and later those that are largely air-borne and seed-borne.

Diseases Caused by Hyphomycetes (Moniliales)

WILT DISEASES (SPECIES OF FUSARIUM, CEPHALOSPORIUM, AND VERTICILLIUM)

The wilts rank among the most deadly of plant diseases, not only because they are killing diseases but also because the wilt fungi often

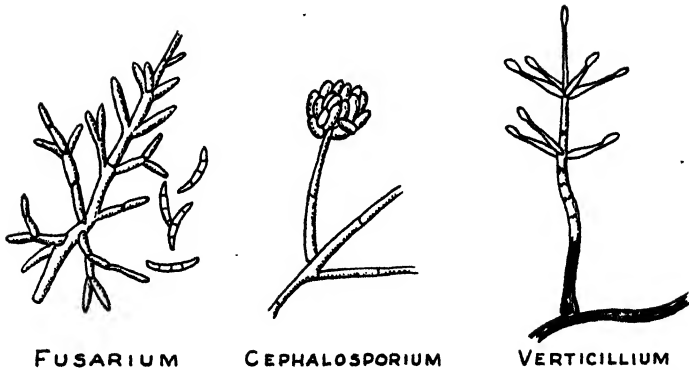


FIG. 67. Three common genera of wilt fungi. *Fusarium* species produce spores of both *Fusarium* and *Cephalosporium* types; *Cephalosporium* lacks the banana-shaped macroconidia but is otherwise like *Fusarium*. (After Wollenweber and Reinking, and Rudolph.)

live for long periods as saprophytes in the soil, and because in general there is no highly effective control measure for them except the breeding of wilt-resistant varieties which is a slow and sometimes unsuccessful procedure.

The principal fungus genera involved are distinguished by the type of spore and its method of production (Fig. 67). All are *Moniliales*, bearing their conidia freely over the surface of the mycelium. In *Fusarium* there are two types of conidia. The larger macroconidia generally are borne in masses (sporodochia) and are banana- or sickle-shaped, clear, with several cells. The smaller microconidia also are clear, usually oval, and from one- to several-celled. Chlamydo-spores—thick-walled swellings of individual cells of the hyphae—are found sometimes, either alone or in rows of from two to several (Fig. 68). The mycelium and spore masses of *Fusarium* may be white, but are often bright colored with various shades of pink, red, orange, blue, and yellow, depending on the species or strain. *Cephalosporium* is identical with *Fusarium* except that it lacks macroconidia. *Verticillium* is so named because of the verticillate or whorled method of branching of the conidiophores. The mycelium is colorless or blackened, the conidia are colorless, and the latter are spherical or egg-shaped and one-celled.

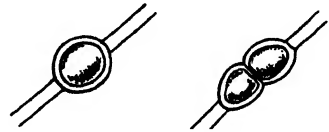


FIG. 68. Typical chlamydo-spores as seen in *Fusarium* species.

COTTON WILT (*Fusarium vasinfectum*)

History and Distribution. There are two fungus wilts of cotton: the *Fusarium* wilt which is widespread in the United States, throughout

the cotton belt, and occurs also in Europe, Africa, South America and India, and the *Verticillium* wilt, first discovered in Virginia in 1914 and since reported from Mississippi, Louisiana, Missouri, Texas, Arizona, New Mexico, Oklahoma, Tennessee, Arkansas, California, Greece and South America. The two wilt diseases produce somewhat similar symptoms in cotton and, although they are not likely to be confused by an experienced observer, they are most reliably distinguished by culturing the causal fungi on agar. Accurate identification is important because *Verticillium* wilt is not controlled by the measures that are effective against *Fusarium vasinfectum*.

Importance. *Fusarium* wilt has long been known as a destructive disease of cotton and was one of the earliest diseases to be combated by the breeding of resistant varieties. Wilt is estimated to cost American growers nearly half a million bales per year, or about 5 per cent of the crop. Individual states report losses of 10 per cent or higher, and on individual farms the loss may reach 60 per cent. Fields may be found in which every plant is killed by wilt before the first picking. The loss percentage of harvested cotton is about three-fifths of the percentage of plants killed by wilt during the growing season since some cotton is usually formed on the plants before they die. The losses are due to reduced yield and lowered quality of the lint harvested from dead or dying plants.

Host Plants. In general the various species of *Fusarium* causing wilts are restricted each to a few host plants. Cotton wilt, tomato wilt, sweet potato wilt, flax wilt, cowpea wilt, and others are caused each by a different fungus, and infested soil that is deadly to one of these crops is safe for the others, so far as wilt is concerned. To a practical extent the cotton wilt attacks only cotton although the closely related okra is susceptible and a few other types of plants have been artificially inoculated with the cotton wilt fungus. Among the various cottons, some of the Egyptian varieties and Sea Island cotton are more resistant than ordinary upland varieties. Thanks to intensive selection and breeding in the past 50 years, high quality wilt-resistant cottons of several types are available now, and more are being developed.

Symptoms and Signs. Wilt is not as readily distinguished from a distance as root rot, since it occurs more commonly in individual plants scattered among healthy ones and not in large, well-defined spots of dead plants. Plants dying of wilt are easily identified by cutting into the wood or peeling back the bark (Fig. 69). The tissues underlying the bark are discolored brown and are often wet, the brown area usually appearing in the form of a ring under the bark when the stem is cut across. The



FIG. 69. Cotton wilt. (*Top, left*) Plant dying of wilt late in the season. (*Top, right*) Healthy plant for comparison. (*Bottom*) Discoloration of the wood under the bark, typical for *Fusarium* wilt diseases. (Courtesy, V. H. Young, Ark. Agr. Exp. Sta.)

discoloration extends up the stem and often far out into the smaller branches. The root system is not decayed, and affected plants are not pulled up easily, as in Texas root rot. In the stage preceding death, affected plants are stunted and yellowed, and single leaves and branches may wilt and die before the entire plant succumbs (Fig. 69). Wilt usually occurs most severely in soils deficient in potash, and the symptoms of potash deficiency or "rust," a browning of interveinal and marginal leaf areas, will often complicate the wilt symptoms on the leaves. The symptom of wilting is not altogether reliable, and may be less commonly seen in the field than stunting, yellowing, and death. The disease typically becomes noticeable between the time the plants are 1 to 2 ft. tall and the first frost, although *Fusarium vasinfectum* is at times also a cause of seedling disease—damping-off—and poor emergence. Under moist conditions, after plants are killed, the bark and adjacent exposed tissues become covered with white mycelium bearing an abundance of macro- and microconidia.

Etiology. *Fusarium vasinfectum* is an imperfect fungus with no well-known sexual reproduction although some related *Fusarium* species have ascomycetous stages. In the absence of a cotton crop the fungus can persist in the soil for several years as a saprophyte or in the form of chlamydospores or conidia. The mycelium invades the seedlings or older plants through wounds in the roots. Within the plant it develops especially in the xylem vessels, interfering with the water supply and ultimately causing death of the plant. The water supply is checked not so much by mechanical plugging of the vessels as by the action of a poison emanating from the fungus. Once the tissues are killed the mycelium grows to the surface of affected parts and produces masses of short cylindrical one- or two-celled microconidia and later four-celled, curved, spindle-shaped macroconidia. These are air-borne but serve mainly to contaminate the soil and seed and not to initiate new infections during the current year. Thick-walled chlamydospores are formed also at tips of, or along, hyphae and these return to the soil with the decomposition of the cotton stalk. The disease is spread from place to place by any means that moves contaminated soil, by drainage or irrigation water, and by air-borne conidia. It may be carried on the seed but there is very little evidence to indicate that infested seed is a common means of introducing the disease into new areas. The fungus does not exhibit true physiologic specialization, but different cultures of it differ in aggressiveness of their attack on cotton. Cotton varieties that are normally resistant may be injured by the more virulent strains.

Epiphytology. *Fusarium vasinfectum* grows between 50°F. and 104°F.

with maximum growth at 77° to 86°F. and maximum infection with shortest incubation period when the soil is at 82° to 90°F., temperatures that are favorable also for growth of the cotton plant. In culture the fungus is relatively independent of variations in pH, developing well from pH 2.5 to pH 9.0, with the best growth, however, in the acid range (pH 3.5). Likewise, the disease is of little importance in neutral soils, occurs rarely if ever in alkaline soils, and is of major importance in acid soils. The occurrence of the disease does not appear to be strongly dependent on particular soil moisture content although the rapidity of the onset of symptoms is conditioned by the amount of water available to the plant. In general, light, sandy soils low in organic matter are more conducive to the disease than heavier soils. Experiments in numerous wilt locations show that wilt often is associated with potash deficiency in the soil, while the addition of potash alleviates the disease. Nematodes also play a part in predisposing plants to wilt. When the root knot nematode is present in the soil, even wilt-resistant cotton varieties, such as Miller 610, may succumb to wilt. Cotton wilt often fluctuates sharply in amount from year to year in the same field. What the principal factor in this fluctuation may be is not clear; possibly winter temperatures or spring rainfall influence these fluctuations to a greater extent than is now realized.

Control. The control of cotton wilt rests on the facts that the causal organism may live in the soil for years, that it is not seed-borne in important amount, that its attack is most severe on plants in potash-deficient soils, and that certain cotton varieties are wilt-resistant. The procedure in controlling wilt depends on the circumstances involved in each case. Where the infestation is not severe, change to one of the better wilt-resistant cotton varieties will suffice. If the plants show signs of potash hunger or "rust," enough potassium-containing fertilizer should be added to correct this trouble. Applications of organic material, such as green or stable manure, are helpful. If the soil is infested with both the wilt fungus and root knot nematodes, wilt-resistant cottons should be grown in a rotation that will control the nematodes, e.g., cotton following two years of cereal or grass crops or nematode-resistant legumes, and use should be made of the wilt-resistant, nematode-tolerant cotton varieties listed below. These control measures do not apply to the *Verticillium* wilt.

The U. S. Dep. Agr. Yearbook of Agriculture for 1936, pp. 682-686, gives an interesting account of the breeding of wilt-resistant cottons, beginning in 1895 with the selections made by two South Carolina farmers, aided and guided by the pioneer plant breeder for disease resistance, W. A. Orton, federal plant pathologist. The selections gave us the variety, Rivers, which was soon followed by Dillon, a systematically obtained

derivative from an Egyptian variety. Apart from its wilt resistance, Dillon had little to recommend it, but when crossed with Dixie, another wilt-resistant selection, it produced such desirable progeny as Triumph, Cook, Dixie Triumph and others. The work on breeding wilt-resistant cottons possessing other good qualities continues today with the efforts of state and federal experiment stations and private breeders in every cotton state. Among the desirable wilt-resistant cottons thus far available and adapted to different regions are Rhyne Cook, Coker 4-in-1, Coker 100WR, Cleve-wilt 6, Wannamaker Cleveland, Early Wilt, Cook 144, Cook 307, Perry Toole, Sykes WR, Delta Dixie WR, Tifton Dixie Triumph, Dixie Triumph 12-5, 25, 55-85, and 62-75, Dixie 14-5, Miller 610, Delfos 425, Deltapine 12 and 829 hybrids, Wannamaker Stonewilt, Rhyne Cleve-wilt, Wilds strains 13, 12, and 11, wilt-resistant Cleveland and Rowden strains, S. and C. Big Boll, D. and P.L. 11A, Tifton Sta. 21, and a wilt-resistant strain of Empire. The first 10 of these and a number of the Dixie strains are tolerant also of root knot nematodes, and do well in soil containing both wilt fungus and nematodes.

TOMATO WILT (*Fusarium lycopersici*)

Fusarium wilt and the nematode disease root knot are two leading tomato diseases in the southern states but neither is of such great importance in the northern half of the United States except under greenhouse conditions. From 10,000 to 30,000 tons of canning tomatoes and upwards of half a million bushels of market tomatoes are lost through wilt each year. Losses in individual states of 10 per cent of the crop are common, and occasionally the state reports reach 20 to 35 per cent of the crop. This of course means that in many individual fields the loss is total.

Tomato is the only known natural host of *Fusarium lycopersici* although successful inoculations followed by injury have been made into onion, freesia, alfalfa, pear, and clover. Tomato species and varieties vary in their susceptibility to wilt. Some strains of the red currant tomato, *Lycopersicon pimpinellifolium*, are immune and are being used in breeding as sources of resistance for the cultivated tomato. Among the cultivated varieties there are many that show moderate to high resistance to wilt. Those in most general use for field and garden planting are Marglobe, Rutgers, Pritchard, and Pan America and, for greenhouse culture, Blair Forcing, Marhio, and Michigan State Forcing. All are high quality, productive types.

Tomato is subject to three other wilt diseases, bacterial wilt, *Verticillium* wilt, and a disease indistinguishable from the common *Fusarium* wilt, but caused by a different species, *F. retusum*. Each of these wilt diseases affects some or all of the tomato varieties that are resistant to the common

Fusarium wilt, and before the use of resistant varieties can be recommended to growers, accurate diagnosis, which sometimes requires isolation of the causal organism on culture media, must be made.

Recent achievements of plant breeding include development of tomato varieties that are resistant to two or more different diseases, among which is the Riverside tomato, resistant both to common *Fusarium* wilt and *Verticillium* wilt.

Symptoms. The symptoms of tomato wilt are similar to those of cotton wilt: yellowing, wilting, dieback, and death of the maturing plant from midseason onward. A cut across the stem shows the darkened vascular ring characteristic of the *Fusarium* wilts. The fungus sporulates abundantly, producing both macro- and microconidia on dying and dead plants. It can live for long periods as a saprophyte in the soil. Infection occurs through the young roots; the xylem tissues are invaded, but the principal damage is due to a toxin excreted by the fungus. All parts of the plant are attacked, even the seed. The dissemination of the disease may occur through either infested seed or seedlings. Tomato wilt develops best at rather high soil temperatures, 82° to 88°F., and is inhibited above 91° or below 70°F. It is favored by a rapidly-growing, succulent condition of the host plant and any factors that stimulate this type of growth, such as high fertility and abundant soil moisture. Acid soil is most favorable. Varieties that are only moderately resistant develop more wilt when soil fertility is either excessively high or low. Races of the wilt fungus of different degrees of virulence occur, and the apparent loss of resistance in some tomato varieties may be due to the appearance of particularly aggressive races of the fungus.

Control. Control of wilt depends primarily on excluding the disease from noninfested tomato plantings by using seed from a healthy crop and wilt-free seedlings. The use of certified tomato plants is a good protection against introduction of the disease into previously wilt-free plantings. Once the soil is infested only the varieties listed as resistant should be used, and in cases of heavy soil infestation it would be better to wait a year or two before using it again for tomatoes. The root knot nematode appears to predispose even wilt-resistant varieties to wilt, and where the nematodes are in wilt-infested soil, a cropping plan should be followed to control them before undertaking the use of wilt-resistant varieties.

WATERMELON WILT (*Fusarium niveum*)

Wilt is by far the most serious disease of watermelons. At various times and in various localities in the past it has led to total abandonment of watermelon culture (Fig. 2). It is so prevalent in all watermelon-growing

sections that most growers sooner or later are faced with the choice of either turning to wilt-resistant varieties, moving the melon plot to a virgin field, or giving up watermelon culture altogether. Fortunately, intensive breeding in a number of states has given the grower a choice of wilt-resistant varieties of various types adapted to the grower's or market preferences, long or round, large or "icebox size," green, gray, or striped, black- or white-seeded, with red or pink flesh, and with or without the hard rind adapted for shipping.

The main features of watermelon wilt are similar to those of the other *Fusarium* wilts. Watermelon is the only important host. Wilts of other cucurbits ordinarily are due to other organisms. The fungus may be carried on the surface of the seed. Once the soil is infested, it can remain so for from 15 to 18 years. The symptoms include seedling blight and, later, wilting, yellowing, and dying of the runners, often one by one. The vascular ring is discolored near the crown, but this discoloration may not be seen farther out in the runners. Macro- and microconidia are produced on the dead vines, and chlamydospores within the infected tissues.

Control. Control consists almost entirely of the use of wilt-resistant varieties. These include the large-fruited varieties Leesburg, Stone Mountain Nos. 5 and 119, Kleckley No. 6, Dixie hybrid, and Blacklee; the medium-sized melons Hawkesbury, Georgia Wilt Resistant, and Blue Ribbon; and the small-fruited Klondike R-7, Wilt Resistant Early Queen, and Black Kleckley. For the best adapted type for each area and purpose, growers should consult their local experiment stations or agricultural extension workers.

Since this and other watermelon diseases may be introduced as spores on seed surfaces, seed disinfestation with bichloride of mercury or one of the organic seed disinfectants is a desirable safeguard. In Tennessee promising results have been obtained in controlling watermelon wilt by spot disinfestation with chloropicrin, the gas being applied only to the hill in which seed are to be planted.

FLAX WILT (*Fusarium lini*)

Flax wilt, the most outstanding disease of this crop, is the reason why flax has always been a pioneer crop, ever moving into new areas as the wilt fungus regularly laid waste the older fields. Until the development by Bolley of wilt-resistant flax varieties which constituted a major turning point in the history of flax culture, there was no alternative but always to seek new land to replace the "flax sick" soil of earlier flax crops. Not only is the crop destroyed, but the oil from wilt-infested flax contains a poisonous substance.

Etiology. The etiology of flax wilt is similar to that of the other wilts, except that *F. lini* is introduced into noninfested fields not only as spores on seed surfaces but also as mycelium within the seed hull. As in the other wilts the disease is recognized by browning of the vascular ring, accompanying wilting and death.

Control. The control of flax wilt rests almost exclusively on the use of wilt-resistant varieties, of which Bison, Linota, Redwing, Buda, Redson, Victory, Sheyenne, Walsh, Biwing, Arrow, and Rio are in common use. The last four varieties named are resistant also to rust—a second major flax disease.

SWEET POTATO WILT OR STEM ROT
(*Fusarium batatatis* and *F. hyperoxysporum*)

This is one of the major diseases of the sweet potato, similar to the other wilts. It is a killing disease recognized by dying of the runners with browning of the vascular tissues (Fig. 70). The development of wilt-resistant varieties has not progressed to the same extent as in the other wilts. Many of the common varieties, such as Yellow Jersey, Red Jersey, Porto Rico, Nancy Hall, Gold Skin, Georgia, and Big Stem Jersey, are highly susceptible, while others are only slightly injured by wilt, including Creola, Dahomey, Haiti, Key West, Pierson, Pumpkin, Red Brazil, Southern Queen, Triumph, White Yam, and Yellow Strasburg. Control also benefits from the following practices:

1. Use of the same land for sweet potatoes not oftener than once in three or four years. It is not safe to use tobacco in this rotation as this crop is susceptible to some of the strains of the sweet potato wilt fungi.
2. Selection of disease-free mother potatoes for propagation, since the disease persists in the roots.
3. Use of vine cuttings instead of sprouts for field planting, or use of certified sweet potato plants that have been inspected and declared free from wilt.
4. Use of sterilized soil for bedding.
5. Dipping cuttings or sprouts in a suitable fungicide, such as suspensions of Spergon or Fermate.

CABBAGE YELLOWS (*Fusarium conglutinans*)

This *Fusarium* wilt, one of the more destructive diseases of cabbage wherever it is grown under warm summer weather conditions, is an outstanding example of success in breeding for disease resistance, beginning with the pioneer work of L. R. Jones in Wisconsin, about 30 years ago,

and continuing with the work of J. C. Walker and his associates in Wisconsin.

As in the case of the other wilt diseases, once soil becomes infested with the cabbage wilt fungus, it is deadly to susceptible types of cabbage. The disease may begin in the seedbed but usually shows in the field two to



FIG. 70. Stem rot or wilt of sweet potato. A plant obtained from the field and cut open in such a way as to show blackened fibrovascular bundles extending from the stem into the roots. The fungus will grow from the stem into the roots in the field and from the potatoes into the sprouts in the seedbed. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

four weeks after transplanting, when the plants look lifeless, turn yellowish green, are stunted, and then die. (Fig. 210.) The vascular bundles of the cut stem show the familiar darkening of wilt diseases. Throughout the season more and more plants may die until destruction of entire fields may be nearly complete.

The disease is favored by soil temperatures above 60° but below 90°F. and, as in cotton wilt, by potassium deficiency in the soil. Soil moisture

and reaction have little effect on cabbage wilt. The disease may be introduced first in diseased seedlings, soil on agricultural implements, cabbage refuse, and surface drainage water, but not on seed.

The disease chiefly affects cabbage, kohlrabi, and most kale varieties, is not of importance on other members of the cabbage tribe, and does not affect plants outside the cruciferae. There are now many resistant varieties of cabbage in general use, including early to late maturing types, and varieties adapted to different areas and purposes. Chief among these are Jersey Queen, Resistant Detroit, Racine Market, All Head Select, Marion Market, Globe, Wisconsin Ballhead, Wisconsin All Seasons, Wisconsin Hollander, Red Hollander, and Bergner. The characteristics of these and a fuller account of the disease are to be found in the reference on cabbage diseases cited at the end of this chapter.

OTHER FUSARIUM WILTS

Fusarium wilts attack numerous other crops. The symptoms and etiology as seen in the preceding cases apply generally. Among the important



FIG. 71. *Fusarium* wilt of potato. Darkening of the vascular ring, indicating invasion of the water-conducting tissues is a common symptom of the *Fusarium* wilts of potato. Tubers such as this should be discarded and not planted.

diseases caused by this group are cowpea wilt, Irish potato wilt (Fig. 71), banana wilt ("Panama Disease," the greatest factor limiting banana production in Central America, with no control known), pea wilt, and wilt of China asters.

CEPHALOSPORIUM WILTS

Cephalosporium is so closely related to *Fusarium* that it may be regarded as a depauperate form of *Fusarium*, lacking macroconidia but otherwise



FIG. 72. *Cephalosporium* wilt of American elm. Note the similarity of symptoms to those of the Dutch elm disease in Fig. 5. (Courtesy, M. A. McKenzie, Mass. Agr. Exp. Sta.)

being identical with *Fusarium*. Species of *Cephalosporium* are implicated in a few important plant diseases, best known of which are the wilt diseases of elm and of persimmon. Both are primarily air-borne rather than soil-borne diseases.

The American and slippery elms are affected by a *Cephalosporium* wilt disease that occurs in most parts of the United States and is important both as a killing disease in itself and because it so closely resembles the Dutch elm disease that culturing is necessary to distinguish the two. Browning of the vascular ring, observed in pencil-sized twigs, is charac-

teristic. *Fusarium*-like microconidia of the *Cephalosporium* stage serve to spread the disease, and less commonly the fungus produces conidia within pycnidia, causing it to be classified as *Dothiorella ulmi* among other pycnidia-forming imperfect fungi.

Control recommendations include: accurate diagnosis by culturing, and eradication and burning of badly infected trees and all dead branches of trees left standing, with disinfection of the cut surfaces.

Cephalosporium wilt of native persimmon (*C. diospyri*) has become prevalent throughout the cotton belt during the past decade, killing out many persimmon groves. Some farmers and ranchers regard it as a beneficial disease, since the persimmon tree is a pest in pastures and often requires laborious eradication efforts. To others, who value the tree for its fruit or very hard wood, the loss of the persimmons is a serious one. The disease is recognized by a blackening of the vascular ring, and the presence of great numbers ("spoonfuls") of pinkish microconidia. The dark vascular ring must be distinguished from similar discolorations due to fire injury, insects, or other causes. The Asiatic persimmon, which is beginning to be cultivated for its fruit, is resistant, but beyond substituting this for the native persimmon, there are no effective control measures. Early fears that the disease might wipe out the persimmon in the fashion of the chestnut blight have not been borne out.

VERTICILLIUM WILTS

Wilts caused by *Verticillium albo-atrum* are common in many species of plants, and while generally less virulent than the *Fusarium* wilts, they often cause serious losses and are important also in complicating the diagnosis of *Fusarium* and *Cephalosporium* wilts. In South America the *Verticillium* on cotton is a major pest, and in the United States and Europe *Verticillium albo-atrum* is fairly common on many plants including shade trees, particularly elm and maple, stone fruits, bush fruits, strawberries, hops, many ornamentals and weeds, and numerous truck crops, including potato, beet, cucumber, muskmelon, okra, peppers, tomato, rhubarb, and watermelon. *V. albo-atrum* often causes a discoloration of the xylem, similar to that due to *Fusarium* species. The symptom of wilting is not as common as yellowing or browning of the leaves, and defoliation, followed by death of part or all of the plant. In cotton a chlorotic marbling of the leaves is characteristic. Sometimes trees recover. Infection takes place mainly from the soil, through the root system.

Verticillium wilt of cotton, in contrast to *Fusarium* wilt, is restricted mainly to highly alkaline soils, and consequently rarely overlaps *Fusarium* wilt. In neutral soils both may be found in the same field with *Fusarium*

active on the sandy, acid spots and *Verticillium* in the heavy soils approaching alkalinity. There are no records of *Verticillium* wilt occurring in acid soils. *Verticillium* wilt of cotton is steadily increasing in the United States, and is a disease to be feared. One infested Texas county reports a 20 per cent loss from the disease. Beyond the variety Acala 1517, which is moderately resistant, only limited progress has been made thus far in developing resistant cotton varieties. Rotation with alfalfa or grain crops is suggested for reducing the infestation.

Verticillium wilt of tomato, caused by the same species of fungus, is principally important in Utah and California and locally in the Atlantic States. The disease resembles *Fusarium* wilt, with darkened vascular ring, but is less severe, stunting but usually not killing the plants. It flourishes at somewhat cooler soil temperatures than favor *Fusarium* wilt. Control chiefly involves a six- to seven-year rotation in which tomatoes, peppers, eggplants, or potatoes are not grown. For California conditions the varieties Riverside and Essary, resistant to both wilts, provide a more effective control measure.

ROOT ROT DISEASES

TEXAS ROOT ROT (*Phymatotrichum omnivorum*)

History and Distribution. Texas root rot, cotton root rot, or *Phymatotrichum* root rot has been known since 1888 as the leading plant disease of the Southwest. Root rot occurs throughout the greater part of Texas except the Texas Panhandle, the adjacent states of Mexico, the Red River counties of Oklahoma, the extreme southwestern corner of Arkansas, the southwestern and southern edges of New Mexico, the southwestern half of Arizona, the southwestern corner of Utah, and the southeastern edges of Nevada and California. (Cf. map of this area in *Arizona Exp. Sta., Bull.* 71, 1937, p. 304.) It has a tendency to follow river valleys in fingerlike projections. The disease has been in this area for at least 50 years and there is little indication that it will involve new areas to a serious extent. Its apparent spread is due to the introduction of susceptible crops in locations that were formerly infested but showed no obvious sign of infestation because they were in native pasture or resistant crops.

Importance. The disease is of utmost importance both because of its destructiveness in leading crops and its wide host range. In all crops the loss in Texas alone is 10 to 15 per cent per year, estimated at \$100,000,000 and about \$50,000,000 loss in the other affected states is likely. Such enormous losses mean that root rot is often the deciding factor in the suc-



FIG. 73. Alfalfa field showing many spots infested with root rot. As the infested areas grow together their roughly circular shape is less apparent. (Courtesy, R. B. Streets, Ariz. Agr. Exp. Sta.)

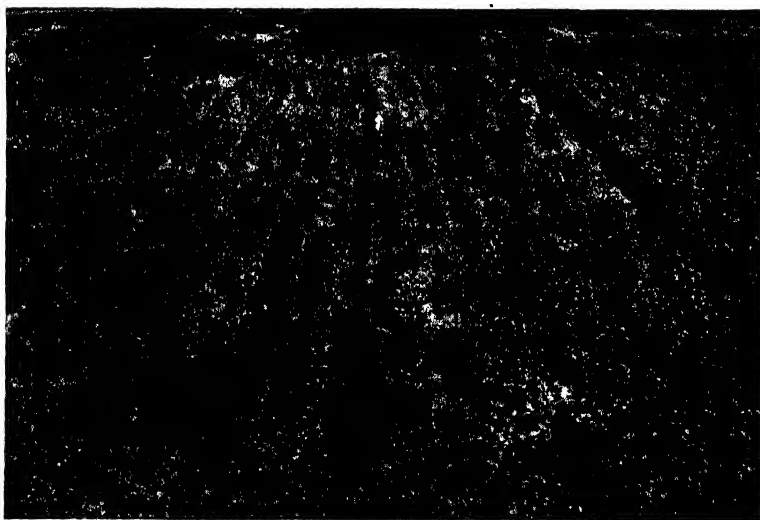


FIG. 74. A large root rot spot in a cotton field. Note that a few plants have survived although surrounded by dead plants. Many trials have shown that the survivors have no resistance to the disease but have accidentally escaped the infection. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

cess of farming in the Southwest and this is true particularly in present-day trends toward irrigation.

Among the types of loss occasioned by the root rot disease are: killing of plants before a crop is produced; later killing, reducing the amount of the crop; lower grade of the harvested crop; difficult picking of cotton;



FIG. 75. Infected cotton root magnified 4 diameters to show strands of the root-rot fungus on surface of root. (Courtesy, R. B. Streets, Ariz. Agr. Exp. Sta.)

restriction of the crops that can be grown on infested land; and lowered market value of the land. Root rot infestation is as significant a property of soil as its fertility, its slope, or its water supply. The percentage yield reduction in cotton is approximately equal to one-half the percentage of plants killed.

Host Plants. *Phymatotrichum omnivorum* attacks more species of plants than any other known pathogen, there being more than 1700 host species.



FIG. 76. Spore mats of root rot fungus in alfalfa field. The outer, younger zone is pure white and the inner zone after a few days becomes filled with a powdery mass of buff spores. (Courtesy, R. B. Streets, Ariz. Agr. Exp. Sta.)

The more important susceptibles are cotton, alfalfa and nearly all other field legumes, all common fruits except black currant and strawberry, and many vegetables, trees, ornamentals, and weeds. Resistance or escape from the disease is seen in all grain and grass crops, annual crops that are grown in fall, winter, or spring but not in the summer, asparagus, cabbage, cantaloupe, celery, cauliflower, cucumber, garden peas, garlic, kale, mint, most squashes, onion, spinach, a few tree species such as osage orange, cedar, hackberry, Kentucky coffee tree, sycamore, and numerous ornamentals. Detailed lists of plants with their reactions to root rot are contained in *Bulletin 527* of the *Texas Agricultural Experiment Station*, and *Technical Bulletin 71* of the *Arizona Agricultural Experiment Station*.

Symptoms and Signs. Root rot occurs from July until frost. The disease kills plants in more or less circular spots ranging from a few square yards to an acre or more in size (Figs. 3, 73, 74). The plants die suddenly, often after having made excellent growth. Just prior to showing severe symptoms the cotton plants have a "fever," or higher temperature than normal, and this can be distinctly perceived by feeling the leaves in early morning. Death occurs within a few days of the first wilting of the plants. In affected plants the whole root system is decayed and the plants slip out of the soil without effort of pulling.

Affected plants show fine, brownish strands of fungus threads (rhizomorphs) sparsely covering the roots (Fig. 75). Under the microscope the

fibers clothing these strands have rigid needlelike sidebranches at right angles to the main fibers, and these structures positively identify the root rot fungus. Under moist conditions spore mats sometimes appear on the soil about diseased plants. These are 2 to 12 in. in diameter, at first snow-white and cottony, later tan and powdery from myriads of spores (Fig.



FIG. 77. Abundant sclerotia formed in soil culture. The soil was carefully washed away to secure a contrasting background. (Courtesy, R. B. Streets, Ariz. Agr. Exp. Sta.)

76). On large roots and tubers are often found numerous small cushion-like sclerotia or resting bodies about the size of a pinhead, at first light-colored, later dark and warty (Fig. 77).

Etiology. *Phymatotrichum omnivorum* can persist for many years in soil as an active pathogen in the roots of plants or in the dormant condition as sclerotia. The rhizomorphs die with the host plant. Overwintering is accomplished in the roots of perennials or winter annuals or as sclerotia. The conidia appear to play no part in the life cycle; they have not been shown capable of germination. Spread of the disease from plant to plant is by growth of the fungus through the soil. There is little indication of dissemination of the disease into new areas outside the general area of infestation. A number of investigators have been unable to produce root rot in soil taken from root rot spots and moved to new locations. Despite the wording of some state quarantines, there is little or no evidence that major dissemination of root rot takes place through nursery stock from infested areas. Insects are not involved in root rot spread, and for some unknown reason the movement of soil by cultivation appears to play no part in the spread of this disease.

The rate of spread from plant to plant is 2 to 8 ft. per month in alfalfa and 5 to 30 ft. per season in cotton and fruit trees. If a root rot spot is staked out and the development of the spot followed for several years it is seen that the spot slowly enlarges for from two to eight years. Then it suddenly or gradually breaks up, leaving most of the spot bearing healthy plants but with a few separated foci of infection, each of which enlarges over the next few years, joining eventually with the others and reforming the original spot (Fig. 78). Breaking up of spots is unrelated to weather or cultural conditions, and there is some evidence that it is due to natural enemies of the fungus or competition in the soil between the root rot fungus and other soil organisms.

Epiphytology. The limited and well-defined geographic area occupied by the root rot fungus leads to the supposition that its distribution is related to ecological factors. Temperature clearly is a limiting factor to the north of the area of infestation, as the disease develops only during hot weather and is inhibited by cold winter temperatures. It appears to be unable to survive north of a line along which the lowest air temperatures are -10°F . When temperatures are favorable, soil moisture usually is the limiting factor. The disease flourishes with moderate moisture and is suppressed under either very dry or very wet conditions. While commonest on heavy, black, waxy, calcareous soils, it is by no means restricted to these but has been found in nearly every soil type in Texas. It is most severe, however, on heavy, alkaline soils of high fertility. The presence

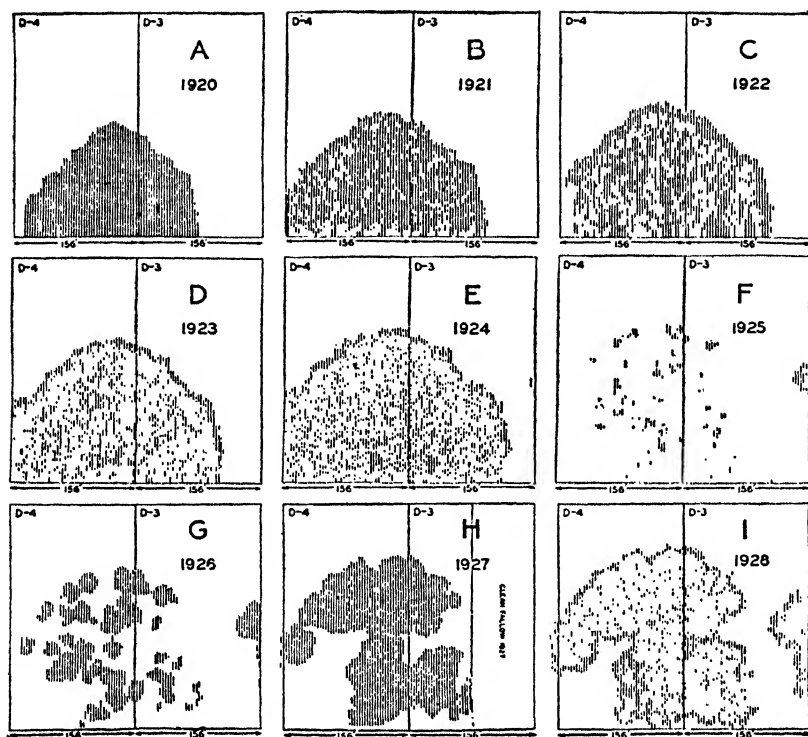


FIG. 78. Breaking up of a root rot spot. From 1920 to 1924 the spot increased in size; in 1925 it broke up and almost disappeared; then it reappeared in 1926 to 1927 with further, less complete breaking up in 1928. The phenomenon is believed to be due to the antagonism of soil saprophytes which at times partially overcome the root rot fungus. (Courtesy, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr., Cir. 173.)

of abundant organic material in the soil lessens the disease incidence, probably through favoring the growth of soil saprophytes antagonistic to the root rot fungus.

Control: 1. **ROTATION.** Standard practice for control of root rot in cotton and other highly susceptible summer crops is to use a three-, or preferably four-year crop rotation cycle in which the susceptible crop is grown one season and oats, sorghum, corn or other highly resistant crops the other years. In Texas extensive use is made of Hubam clover, a winter crop that may be harvested for hay or seed and then plowed under, followed by cotton. This greatly reduces root rot losses.

Such rotations will not rid the soil of the fungus, but will reduce root rot losses to a low level which will not seriously handicap the grower. While this may seem like a long rotation, it is entirely practical and fits in well with the federal crop control program which ordinarily does not per-

mit more than 25 per cent of cultivated land to be in cotton any given year.

2. **AVOID SUSCEPTIBLE PERENNIALS.** Perennial crops rated susceptible will generally result in failures on root rot-infested soil. The prudent grower will not attempt to grow alfalfa, susceptible orchard crops or susceptible woodlot or ornamental trees or shrubs in root rot areas. To determine whether the root rot fungus is present in soil, a susceptible indicator crop, such as cotton or field peas, may be grown in the season before establishing permanent plantings of susceptible perennials.

3. **ERADICATION AND BARRIERS.** Many direct methods for ridding soil of the Texas root rot organism have been tested, some with excellent results. These include chemical treatments of the soil to kill the fungus or make the soil uncongenial for its development, deep tillage, and the addition of large quantities of organic fertilizers. Under most conditions, it is doubtful whether any of these will justify the expense involved on a field basis. Walling off the spots with trenches or barriers as of several rows of sorghum, aids to keep the spots from enlarging, and this is of value in some instances (Fig. 79).

The use of organic fertilizers has special interest because here practical use is made of biological control: the inhibition of the root rot fungus by antagonistic organisms in the soil. Ordinarily soil saprophytes are better adapted to a saprophytic life than plant pathogens that occasionally live as saprophytes. King and Loomis in Arizona have demonstrated almost perfect control of root rot in irrigated cotton by heavy applications (15 to 30 tons per acre) of manure or spoiled alfalfa hay (Figs. 80, 81). They inter-



FIG. 79. Peach orchard showing effective use of barrier to stop advance of root rot through the soil. When barrier was placed between the two rows one year previously, all trees in the left hand row were alive. Root rot advancing from the infested area at left killed trees not protected by barrier. (Courtesy, R. B. Street, Ariz. Agr. Exp. Sta.)

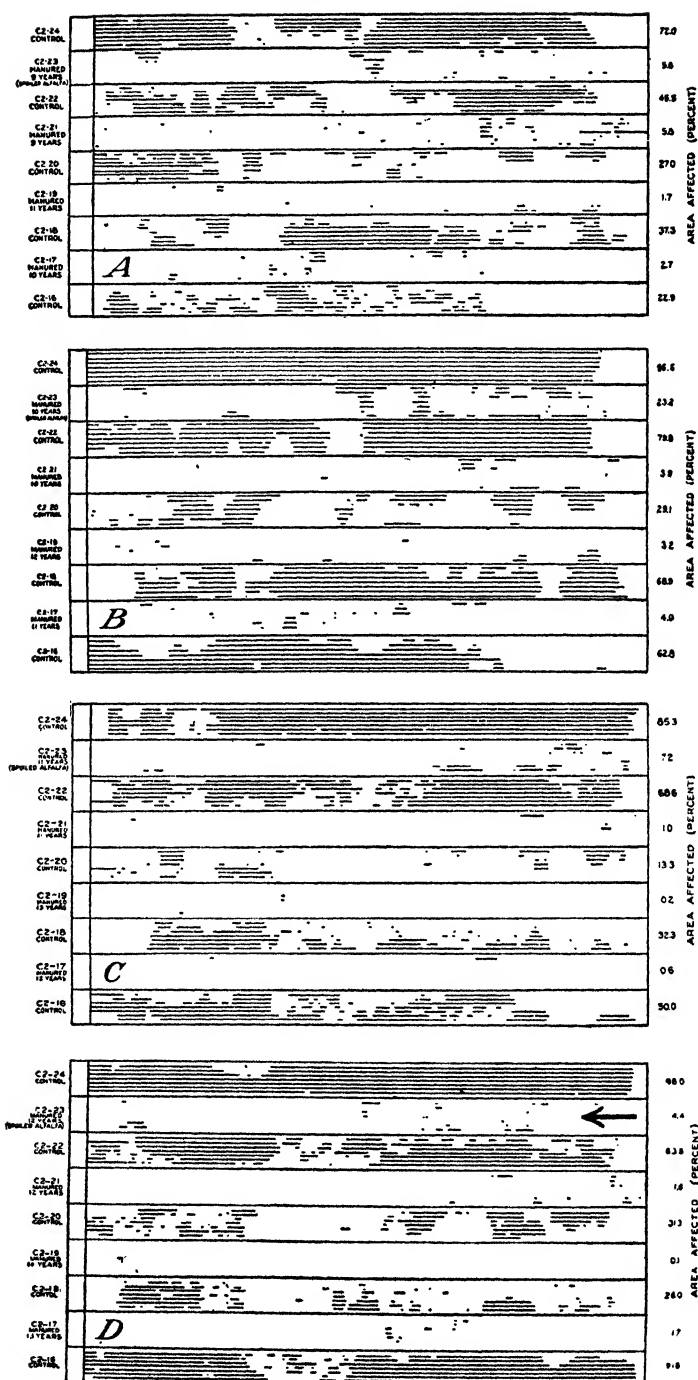


FIG. 80. For legend see opposite page.



FIG. 81. The effect of manuring in controlling root rot. A view of the plots diagrammed in Fig. 80 taken from the position indicated by the arrow in that figure. The center plot above has been manured with spoiled alfalfa hay for 12 years. Unmanured plots are at each side, with manured plots at their outer edges. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

pret this control as due to the antagonism of the soil saprophytes, favored by the humus, against the root rot organism. The increased fertility of the treated soils is not a factor in this means of control, since applications of commercial fertilizers to give equal availability of nutrients fail to effect the control obtained with the organic material.

4. WEEDS. Many weeds are susceptible to the root rot fungus. In any rotation program every effort should be made to keep the land and fence rows free from weeds, as they can easily defeat the purpose of the rotation. Among the most susceptible weeds are: bindweed, cocklebur, dock, ground cherry, horse nettle, jimson weed, lamb's quarters, prickly lettuce, milkweed, ragweed, common sunflower, Russian thistle, goldenrod, wild aster, and spurge nettle.

5. ORNAMENTALS AND FRUIT TREES. Resistant plants should be selected as far as possible. Sometimes affected trees may be saved by pruning back, making a circular ridge of the diameter of the plant top about the tree, and applying ammonium sulfate (1 lb. to 10 sq. ft.) to the

FIG. 80. Chart showing the effect of manuring in controlling Texas root rot. (A, B, C, D) Representing the same plot during four successive years, 1932 to 1935. Shading indicates root rot occurrence in cotton. A photograph taken at the position of the arrow is reproduced in Fig. 81. Note also the breaking up of root rot spots, e.g. in the top strips of B and C. (Courtesy, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr., Cir. 425.)

soil, followed by 3-in. to 4-in. irrigation. The treatment may be repeated after 5 to 10 days, but not again the same season.

6. **VEGETABLES.** Resistant vegetables should be selected as far as possible. Susceptible vegetables will escape injury from root rot if harvested before hot weather.

BLACK ROOT ROT OF TOBACCO (*Thielaviopsis basicola*)

Distribution and Importance. The black root rot disease of tobacco and other crops is widespread in all tobacco growing regions of the United States, Canada, and Europe, and occurs also in Japan and Australia. In the majority of sections it is the most serious tobacco disease. An early estimate placed the loss for the United States at from 10 to 20 million dollars annually, and as recently as 1945, when half the tobacco acreage in Kentucky was planted with varieties resistant to black root rot, a season favorable to the disease resulted in a loss in that state of \$15,000,000.

Suscepts. While tobacco is the main host, the disease has a wide host range, affecting about 100 species of plants, chiefly in the tobacco, legume, and cucurbit families. Many commercial legumes are susceptible, including soybeans, clovers, lespedeza, peanuts, cowpeas, alfalfa, peas, and beans. The black root rot fungus attacks cotton, notably in Arizona, where it causes a purplish dry rot of the tap root; here the disease is confined mainly to seedlings and is not widespread nor greatly damaging.

All of the older commercial types of tobacco are susceptible, but excellent progress has been made in developing suitable high-yielding resistant varieties. This work in Kentucky has been particularly successful with production of strains Ky. 16, 19, 22, 33, 34, 41A, and 52, of different types and uses. Ky. 33 and 34 are resistant also to *Fusarium* wilt and Ky. 34 and 52 are resistant to mosaic. Connecticut has resistant strains of Conn. Broadleaf, Cuban, and Little Dutch. For Massachusetts there is Havana Seed 211 which is being improved. Ten years of breeding work in Virginia has resulted in a number of valuable resistant strains. In North Carolina there have been obtained very resistant strains of Paris wrapper and Jamaica varieties of high yield and good quality, and the resistant Wis. Havana 142 is now being grown on most of the Wisconsin tobacco acreage. Some of the resistant varieties owe their resistance to a good ability to form wound cork behind the lesions, isolating the infected tissues from the healthy.

Symptoms. This is primarily a root disease attacking plants at any growth stage, causing a blackening and decay of taproot and lateral roots. If the plants are young, they die in the fashion of damping-off. If older, they are stunted, with slow growth, which growers sometimes mistake for

the effects of poor weather. The decay proceeds until nearly all roots are destroyed. New adventitious roots may form but these, too, fall victim to the decay. If nitrogen is plentiful in the soil, affected plants are abnormally dark green, and if it is low, they are pale and uneven in color. Affected fields are patchy in appearance with many dead plants, and the living ones of various size and appearance (Fig. 82). Those plants that survive may make a fair late crop if in sandy soil; in heavier soil they are permanently stunted and make few marketable leaves. It may be necessary to reset the whole field once or even twice before a fair, but undesirably late, stand can be obtained.

Etiology. Black root rot is caused by the imperfect, soil-dwelling fungus *Thielaviopsis basicola*. This is often accompanied by an ascospore-bearing organism, *Thielavia basicola*, but there is no reliable proof that the



FIG. 82. Tobacco field laid waste by black root rot which has killed or stunted growth of most of the plants. (Courtesy, F. A. Wolf, Duke University.)

ascospores belong to the root rot pathogen. *Thielaviopsis basicola* reproduces and persists through the formation of conidia and chlamydospores. The conidia are unusual in that they are produced inside conidiophores and forced out one by one through a break in the tip of the conidiophore endwise until 40 to 160 spores may be ejected from one conidiophore. Spores produced in this fashion are termed endoconidia (cf. sweet potato black rot). The chlamydospores are dark, compound, club-shaped, eventu-

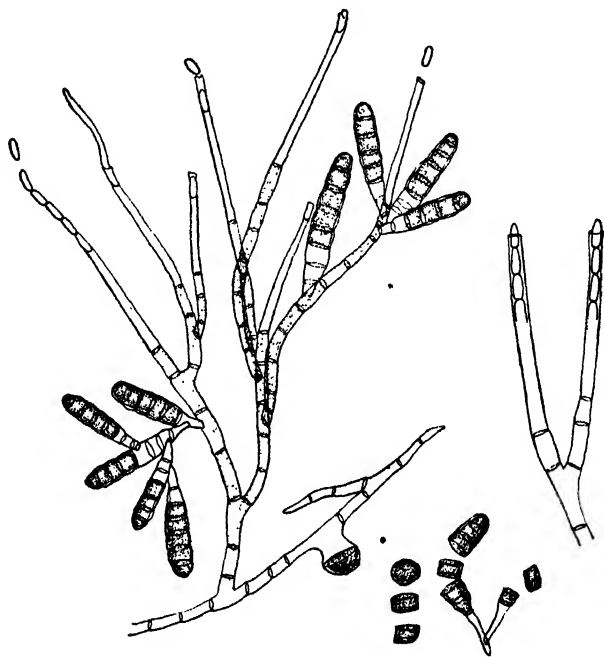


FIG. 83. *Thielaviopsis basicola*, cause of black root rot of tobacco. (Left) Mycelium producing endoconidia and chlamydospores. (Right) Endoconidiophore, enlarged, showing formation and ejection of endoconidia. (Bottom) Chlamydospores breaking up into segments. (After Gilbert, 1909.)

ally breaking up into short cylindrical or pillbox-shaped cells, each of which acts as a spore (Fig. 83). Either endoconidia or chlamydospores can germinate to produce much-branched brownish mycelium. The brown color of mycelium and chlamydospores and the manner of spore production, freely over the mycelium, places this fungus in the group of sooty molds or *Dematiaceae*, many of which are important pathogens and will be considered in the following pages.

The fungus occurs widely in soils as a saprophyte and pathogen of many hosts. It is disseminated by any means of moving soil, and particu-

larly is spread from seedbed to field in diseased transplants. It is not well adapted to air-borne dissemination. There is some indication that infection of roots may be aided by the prior attack of the root lesion nematode (p. 364).

Epiphytology. Black root rot is most destructive in heavy, cold, slightly acid to alkaline soils, well supplied with humus, with excessive water and poor ventilation. It is most active at soil temperatures between 63° and 73°F. and does not do much damage in soil warmer than 79°F., at soil pH below 5.6, or in sandy soils that are low in organic matter. A long wet period after transplanting is particularly conducive to attack, and drought or high temperature may check the disease, leading to late recovery of affected plants.

Control. Precautionary measures are necessary in both seedbed and field. The seedbed must be maintained free of the fungus by use of new forest soil or by disinfestation of the seedbed or a combination of the two. Sterilization of the soil, which can be accomplished with steam or tear gas (chloropicrin), must be deep and thorough. In the field, wet soil should be avoided, and plants from a diseased bed should not be used unless the field soil is very acid. If lime is used, it is best to apply it to an alternating crop in the rotation, not directly to the tobacco crop. Unless resistant varieties are grown, a three-year or longer crop rotation is advised, with cereals rather than legumes in the nontobacco years.

The most effective control measure is the use of resistant varieties, some of which are listed above, with new, improved ones constantly being produced.

DRY-LAND FOOT OR ROOT ROT OF WHEAT (*Helminthosporium sativum*)

Dry-land foot rot commonly affects wheat, rye, and barley, and may be found also on certain grasses, such as quack grass, Kentucky blue grass, wild oat grass, wild rye, and mouse barley. The term "common root rot" is applied collectively to the *Helminthosporium* disease and root rots caused by other, undetermined species of *Helminthosporium* and *Fusarium*. The expression "foot rot," as distinct from root rot, refers to the stage in which the lower inch or two of the stem is discolored and eventually may decay or cause failure to head or sterile heads. *Helminthosporium sativum* is only one of many fungi that attack the roots and stem bases of wheat plants. The discussion here, however, is limited to the disease caused by this fungus.

Importance. The foot rots cause substantial losses in the wheat crop from Texas to Canada. For example, in Kansas alone the loss from all foot

rots in wheat was 4.5 per cent of the crop (11,901,000 bu.) in 1931, 4.5 per cent (5,943,000 bu.) in 1932, 6 per cent (4,727,000 bu.) in 1935, and 5 per cent (9,104,000 bu.) in 1937. The average loss estimates for the United States usually range from 0.5 per cent to 1.5 per cent of the national crop. These are probably underestimates since in Manitoba, where foot rot is probably no more destructive than in many United States wheat areas, systematic surveys have shown that for the three years, 1939–1942, the average yield reduction was 12.1 per cent, representing \$3,827,000 per year. In individual wheat fields that are heavily attacked, from 30 to 70 per cent or more of the plants may be killed before harvest. The greater part of these losses are due to *Helminthosporium sativum*, and one should not forget that wheat is only one of several crops attacked by this fungus.

The loss from foot rots is of several types. Young seedlings may be destroyed, giving a thin stand or necessitating replanting which is a twofold loss, consisting of the time, labor, and seed in replanting, and the fact that a replanted crop is rarely planted at the most favorable time, resulting in a subnormal crop. As the crop approaches harvest the damage from foot rots becomes most apparent, as the plants in large areas of the field die and blacken without setting grain, or produce weak, sterile, or partially filled heads with black point or shriveled grain. Many of the plants which survive and produce are handicapped by a partial loss of roots, with a consequent subnormal yield.

Symptoms and Signs. Foot and root rots have a tendency to appear in the field in well-defined, round, oval, or irregular spots. This may appear in the seedling stage of the disease but in the case of dry-land root rot it is most pronounced at maturity of the crop when the great spots of sterile plants, overgrown with black, saprophytic molds, stand out in striking relief against a golden background of ripened grain. The spots may vary from a few square feet to an acre or more in size. They are not restricted to either low or high parts of the field, but do have a tendency to be more abundant at the edges of the field, possibly indicating spread to the field from fence row grasses or volunteer grain. The spots may be distinguished from spots of poor fertility by the sharp delimitation of the root rot spots, the diseased plants being abruptly bordered by healthy ones, while in the case of poor or alkaline soil, the stunted plants show progressive stages of improvement as one passes from the center of the spot to nearby healthy grain.

The *Helminthosporium* seedling blight may be distinguished by the dark brown lesions of the cortical tissues, lesions that frequently extend into the coleoptile and seedling leaf. This results in dead plants before

or after emergence from the soil. This phase of the disease is said to be more common in the spring wheat than in the winter wheat area.

The fungus continues to cause a rotting of the roots of surviving plants. In less severe cases dark brown lesions develop on the roots at points of infection, causing little apparent injury to the plant. The coleoptile, basal leaf sheath, and stem may show various degrees of infection from small elongated lesions to a complete rotting. In severe cases of root or foot rot the seedling leaves show a dark green discoloration or sometimes a chlorosis and later the whole seedling usually dies. In the case of older plants the type of root and culm injury is much the same as that occurring on seedlings, except that there seems to be less tendency for a complete rotting of the stems.

The fungus attacks the leaf blades, sheaths, upper culm, and head. The kernels also become infected and develop a dark brown to black tip at the germ end (black point) (Fig. 84). So far as known, no part of the wheat plant is immune from infection by this parasite during its development. Lesions of stem and foliage tend to be elliptical in outline. Usually these consist of a dark brown margin with a lighter brown center. On leaves, these centers frequently drop out, and in some cases lesions may involve the entire width of the leaf.

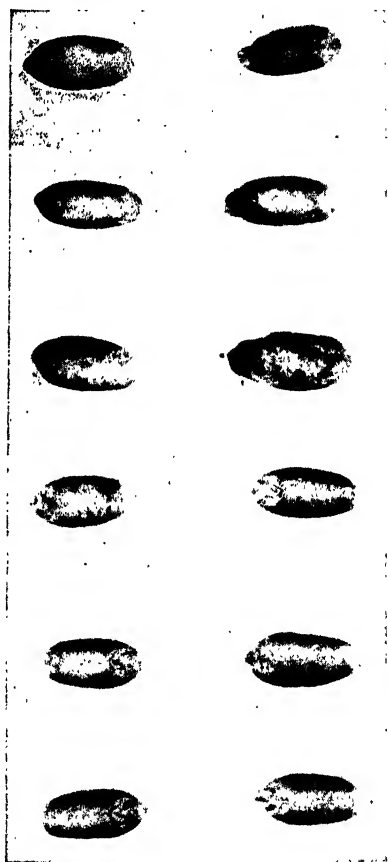


FIG. 84. (Top) Black point or internally infected wheat grains. (Bottom) Healthy grains.

On the roots and stems of wheat seedlings *H. sativum* causes the tissues to develop a light chocolate brown discoloration which can easily be confused with discolorations due to other root rot organisms. The light brown discoloration of the lower inch or two of the culm is characteristic of the foot rot phase of the disease (Fig. 85).

Dark masses of *Helminthosporium* spores are not often seen on affected plants, although the mycelium and spores of secondary sooty molds, over the heads and leaves, are very typical of the disease at harvest time.

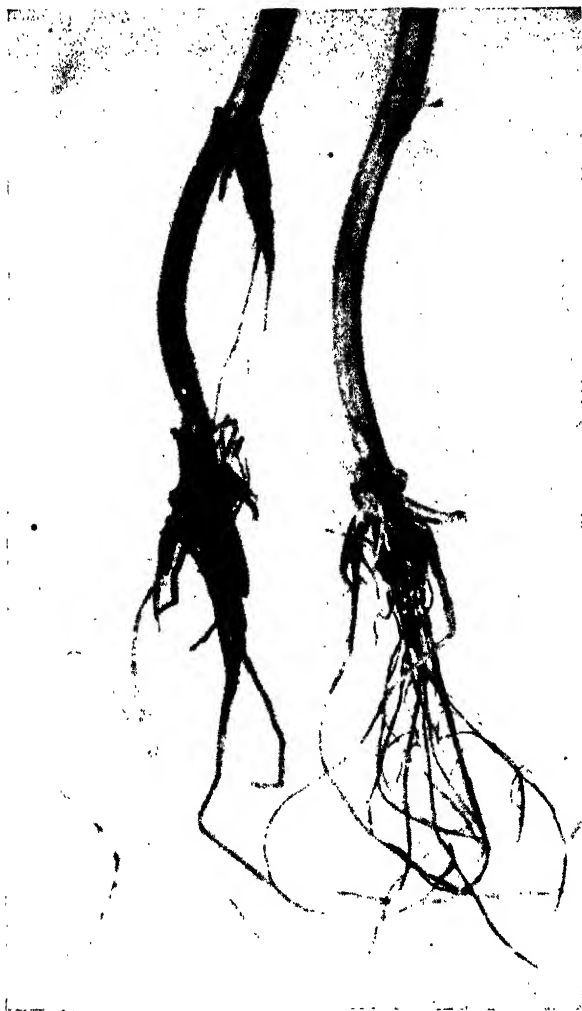


FIG. 85. Foot rot of wheat. (Right) Basal joint of wheat stem slightly affected, showing dark streaks. (Left) Severely affected stem with lower joint darkened instead of white as in the normal stem.

Whitened, shriveled heads also are commonly the consequence of root rot infection.

Etiology. *Helminthosporium sativum*, like *Thielaviopsis*, belongs to the family *Dematiaceae*, or sooty molds, with dark-colored mycelium and conidia. The conidia are produced here and there on mycelium over the surfaces of affected tissues and not in fruiting bodies. The conidia of *Helminthosporium* are large for spores, several-celled, wormlike, and are borne successively on the tips of dark conidiophores which often pro-

trude through stomata of the host plant (Fig. 86). *Helminthosporium sativum* is a facultative saprophyte and grows well on nutrient media and in soil debris. It survives between crops as spores or mycelium either in the soil and crop residue or in the black point seed. Conidia are produced from the saprophytic mycelium in the soil debris, which may initiate the early infections, or the roots of young plants may be invaded directly by the soil-borne or seed-borne mycelium, with consequent rootlet decay. Ultimately, spores are carried from the soil or affected bases of plants to the leaves, where they germinate and produce the first leaf lesions. The leaf lesions produce successive generations of conidia which are air-borne to other leaves and heads and serve to multiply the disease during favorable weather conditions. Many of these spores carry the infestation back to the soil. At the same time there is an onward march of the mycelium through the soil, continually enlarging the spots of infection. The many cycles of infection continue till maturity of the plants and the production of black-tipped kernels in affected heads. After harvest the soil is further contaminated by mycelium and spores in the stubble, straw, and chaff.

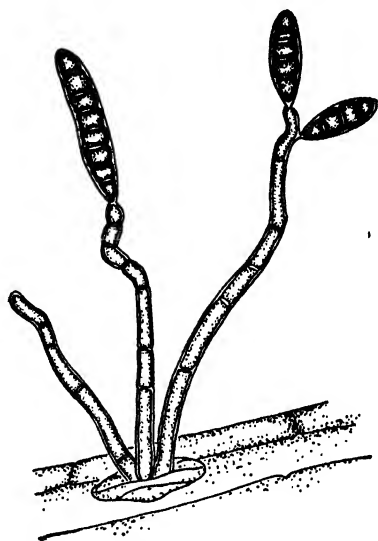


FIG. 86. Conidia and conidiophores of *Helminthosporium*, emerging from a stoma. (After Dreschler.)

The part played by other hosts should be remembered. *H. sativum* may include in its life cycle infections of rye, barley, and certain grasses, and these aid in the survival of the fungus between wheat crops and in increasing the inoculum during periods in which wheat and these other hosts are growing together.

H. sativum is not a highly specialized parasite, as seen by its saprophytic life in the soil, and the fact that it attacks weakened hosts in preference to vigorous ones. Christensen has shown that the fungus includes as many as 37 types or races, differing physiologically and structurally. These variants also show differences in pathogenicity, corresponding to an indefinite type of physiologic specialization, in which some races are highly virulent on wheat and barley, others much less so (Fig. 87). He has shown that new races may arise through nonsexual mutation in pure cultures and that these may in time revert back to the parent form (Fig. 88).



FIG. 87. Variation in *Helminthosporium sativum*. The pair of pots at the extreme right contain wheat plants in uninoculated soil. The other pots contain soil inoculated with various strains of *H. sativum* showing (l.f. to right) decreasing degrees of virulence. (Courtesy, J. J. Christensen, Minn. Agr. Exp. Sta.)

Epiphytology. *H. sativum* is tolerant of a rather wide range of temperature although it is favored by warmth. The mycelium will grow at any temperature from 34°F. to 99°F. with an optimum near 82°F. Likewise, the spores will germinate from 43°F. to 102°F., with optimum germination between 72°F. and 90°F. Infection can occur from 54°F. to 93°F. but is most severe at 72°F. to 93°F., and the disease develops most rapidly at the higher temperatures. Spores can withstand exposure to both low and high temperatures for considerable periods of time if the relative humidity is low. They can also withstand long periods of alternate freezing and thawing, and can overwinter as far north as Minnesota, although the percentage of surviving spores differs in different seasons. Within seed the mycelium is killed by heating the seed to 203° to 212°F., dry heat, for 30 hours.

High soil moistures favor root infection, especially at high temperatures. The moisture optimum varies with the temperature. Extremely low soil moistures are unfavorable for infection at all temperatures, and at extremely low or high temperatures the moisture curves for infection are very irregular. The ill effects of root infection are more severe in extremely dry and extremely wet soils than in soils containing an optimum amount of moisture for the growth of the plant. Leaf and spike infections increase directly with the amount of moisture present, and are most abundant following heavy dews and rains.

It is noticeable that the environmental conditions favoring growth and reproduction of the parasite are not necessarily those associated with the most severe disease. This is due largely to the fact that the environment also is operating on the wheat plant, and any factor that weakens the wheat increases its susceptibility to root rot, regardless of whether or not

that factor at the same time favors the development of the fungus. Many such factors might be cited, for example, unfavorable temperature or moisture for wheat growth, competition due to overseeding or weeds, unsuitable soil, and attacks of insects or other diseases. Since the fungus is tolerant of a wide range of environmental conditions, any analysis of the effect of environment on this disease will be largely concerned with the effect of environment in reducing host vigor and thus predisposing the host to the disease. Wheat plants are most susceptible to *H. sativum* from the milk stage on, and this in turn may be correlated with their dwindling metabolic activity as maturity approaches.

In summary, *Helminthosporium sativum* has a tolerance for a wide range of environmental conditions. Herein lies the reason why the dry-land foot rot, unlike such diseases as rusts or downy mildews, is constantly with us, rarely if ever developing in epiphytotic proportions, but each year eating away at the crop and causing losses which vary to only a limited extent from one year to another.

Control. As is generally true of soil-borne diseases, crop management is of first importance in controlling dry land foot rot. Fellows, a leading authority on root rot, advises:

It may be controlled effectively by the proper date of planting. It happens that the date recommended by the agronomist as the best for yield is the

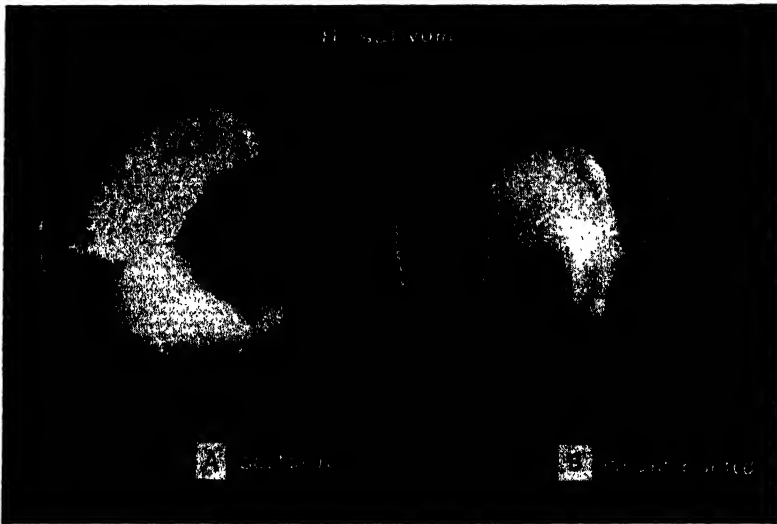


FIG. 88. Mutation and reversion in *Helminthosporium sativum*, cause of dry land root rot of wheat. The agar plate (A) was planted with a pure strain of the dark fungus. As the culture grew it mutated producing two or three white variants. When one of these was planted (B) it exhibited a reverse mutation, reverting back to the original black type. (Courtesy, J. J. Christensen, Minn. Agr. Exp. Sta.)

proper date for planting to avoid foot-rot. I have found this true time and again from one end of the wheat belt to the other. The agronomist worked out disease control without knowing it. In general it is a retarded date as compared to that which the farmers ordinarily practice. I avoid telling a farmer to plant late for the proper date really isn't late but retarded for him. The farmer usually doesn't like a retarded date since it may permit blowing of the soil. In Montana the proper date is around September 12 and in the Panhandle of Texas about October 15, with intervening dates in the intervening territory.

This refers to winter wheat. In spring wheat the disease is avoided by planting as early as good agronomic practice permits.

The planting date is dictated also by many other factors such as available moisture, wind erosion, and avoidance of wireworms. Hence advantage must be taken also of other measures which aid in root rot control.

It has been shown that the weakest plants are the most susceptible to root rot. This implies that any steps which build up the vigor of the crop will at the same time fortify it against root rot. Thus, application of phosphate fertilizers in the drill row in localities where moisture conditions permit, will promote rapid growth of the plants and the early establishment of a properly functioning root system, when this is most urgently needed in overcoming attacks of parasites. In other cases the fertilization should follow the advice of soil specialists aiming at the balance of fertility that is most conducive to normal wheat development. Such fertilization does not prevent the disease from occurring; it appears to act as a means of control by stimulating plants in infested areas to a more vigorous development.

Proper depth of planting also is a factor. When seed is sown at a depth greater than 2 or 3 in., the seedlings are weakened and rendered more liable to attack by root-rotting fungi, both before and after emergence, while seedlings from shallow-sown seed are unable to establish themselves in time to forestall root rot attack.

So far as is feasible, wheat fields and adjacent borders should be kept as free as possible of wild grasses and weeds. The grasses serve as reservoirs of root-rotting fungi, while weeds will compete with the grain for water, light, and nutrients, and predispose the grain to root rot by lowering its vitality. As the wheat loses its vigor from foot rot disease, the weeds flourish, intensifying the trouble as the season progresses. It is inadvisable also to sow wheat on land directly after it has been in grass. If any grain crop must be used in this way, oats is preferable, but it is better to put the land into flax or some other nongramineous crop for a few years after plowing the native or tame pasture.

Crop rotations including legumes or some other noncereal crop will help to lower the concentration of cereal foot-rotting fungi in the soil.

Such plants, together with summer fallowing, tend to starve the root-rotting fungi and allow other soil organisms to hasten their destruction. Oats is probably the least susceptible cereal crop to include in a rotation. In North Dakota tests, wheat after corn or wheat after oats after fallow reduced root rot losses to a moderate level. Barnyard manure or green manure reduced root rot, but only in years of abundant precipitation.

Seed treatments are not specific for root rot but materially decrease the fungus load on infested seed, and also give some protection against soil-borne infection. The dusts used for bunt control are helpful in this respect. Black point seed, however, is internally infected with *Helminthosporium*, and the surface treatments will not control internal infections. These can only be eliminated by selecting for planting purposes seed that is free of black point kernels.

There is little evidence on record which will help in combating root rot by the use of resistant varieties, although most investigators have observed variation in the reaction of different wheat varieties to root rot. The best adapted varieties in each region will make the most vigorous growth and thus be more resistant than more poorly adapted varieties.

The natural microflora has a marked inhibitory action on the growth of *H. sativum* in soil. Bacteria and fungi isolated from the soil show a suppressive action on the pathogen. Although *H. sativum* sporulates well in certain sterilized soils it will not if the soils are unsterilized. The evidence suggests that sporulation is inhibited by saprophytic soil organisms. The fact that root rot diseases of wheat are less severe when the crop is grown on fallowed land than on land cropped to wheat for several years, may be related to the growth of soil saprophytes, which in bare fallow have an advantage over the pathogens in competition for food. Whether or not this natural control can be used directly in controlling root rot, as has been done with Texas root rot and the pineapple nematode, in any case our knowledge of this natural control aids us in understanding the disease and its control by cultural practices.

LEAF, SHOOT, AND FRUIT DISEASES

HELMINTHOSPORIUM DISEASES OF BARLEY

Barley is attacked by five species of *Helminthosporium*. Three of these, causing stripe, spot blotch, and net blotch are common and at times quite destructive. Each of these diseases has its own peculiarities in etiology, epiphytology, and control.

Barley stripe, caused by *Helminthosporium gramineum*, is a systemic disease with life cycle and control very much like that of bunt of wheat.

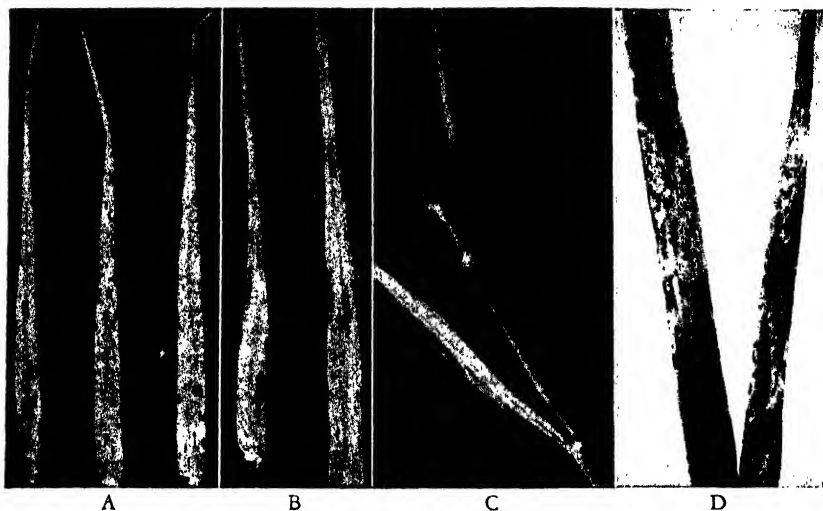


FIG. 89. *Helminthosporium* diseases of cereals. (A) Barley spot blotch (*H. sativum*). (B) Barley net blotch (*H. teres*). (C) Barley stripe (*H. gramineum*). (D) An uncommon *Helminthosporium* spot disease of wheat growing in a very humid environment.

Affected plants show white or yellow streaks on the leaves which later split into dry brown ribbons coated with dark mycelium and conidia (Figs. 89, 90). The plants usually die before harvest, without producing grain. The conidia may be borne to healthy barley heads where the fungus survives as mycelium between the hull and seed coat. When such a kernel is planted, the mycelium grows into the young seedling and spreads

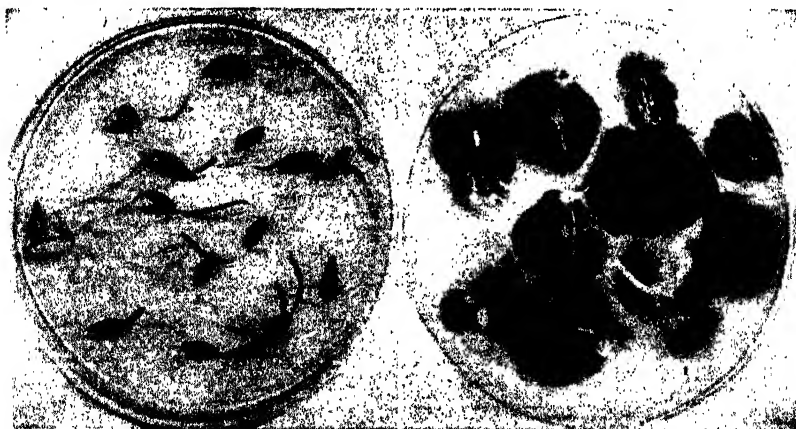


FIG. 90. Germinating barley seeds. (Left) Healthy seeds. (Right) Seeds infected with *Helminthosporium* blight, showing the black mycelium growing out on the agar from each seed. (Courtesy, W. E. Brentzel, N. D. Agr. Exp. Sta.)

through the developing plant, destroying it at or before the time normal plants are heading. Stripe does not survive in the soil to any great extent, and does not spread from the leaves of one plant to those of another during the growing season. It is favored by cool temperatures (under 68°F. at planting time). Effective control is obtained by dusting the seed with a volatile disinfestant dust similar to that for the control of the seedling-infecting types of wheat and barley smut. Lists of barley varieties with resistance to stripe are given in *Phytopathology*, **34**, 572-585 (1944), and **36**, 547 (1946), and in the *Journal of the American Society of Agronomy*, **35**, 736-737 (1943).

Barley spot blotch is caused by *Helminthosporium sativum*, the same fungus that causes dry-land foot rot of wheat. The disease commonly is recognized as oval, chocolate-brown, uniform, well-defined leaf spots, followed by dieback of the leaf tip. The plant is injured by reduced food-making ability. Seedlings often are attacked and destroyed by a serious seedling blight due to the same fungus, favored by warm, wet soil. A foot rot, comparable to that of wheat, represents a third form of attack on barley. Grains of affected plants are sometimes internally infected, recognized as black point or one form of "blighted barley." The fungus survives in the soil where it may first attack barley roots, or the first infections may result from planting "blighted barley." In the growing season the disease spreads from plant to plant by air-borne conidia produced on infected leaves or in soil debris. The fungus is tolerant of wide environmental variation but is favored by high temperatures and ample moisture. Control depends on early, thorough cultivation to decompose crop residues, and a two- or three-year rotation with crops other than grain, best including one soil-building crop. Blighted barley should not be used for seed. Seed dusting, as for smut, is an accessory aid. Some barley varieties, such as Manchuria, Minsturdi, Nepal, Glabron, Peatland, Velvet, and Minn. 184, show resistance to spot blotch.

Barley net blotch (*Helminthosporium teres*) rarely affects seedlings or roots but is primarily a leaf disease, causing elongate, spindle-shaped dead spots with a netlike or ladderlike pattern of dark veins seen when the leaf is held up to the light. The leaf dies back to the affected areas, and yields are reduced as a result of lowered photosynthesis. Affected kernels sometimes show light-brown hull discoloration but usually are not discolored at the base. This is one of the *Helminthosporium* species which forms ascospores, in perithecia on old stubble, and these are an important source of spring infection. In contrast to spot blotch, net blotch is favored by cool temperatures, 50° to 60°F., and net blotch usually is replaced by spot blotch as the warm season advances. Net blotch damage is reduced

by deep and thorough plowing to aid decomposition of crop residue, a two- or three-year rotation using any crop other than barley, seed dusting as for smut, and avoidance of "blighted barley" as seed. No data are available on varietal resistance.

OTHER HELMINTHOSPORIUM DISEASES

Corn in America is attacked by five species of *Helminthosporium*. Three of these are of minor significance. The other two, *H. turcicum* and *H. carbonum*, cause leaf spot followed by premature drying up of the leaves, with reduction in fodder and ear yields. *H. turcicum*, which causes long elliptical greenish-brown lesions, attacks both field and sweet corn. It regularly causes some injury from Indiana and Ohio eastward, and in 1942 it was epiphytotic in this area, resulting in serious losses. *H. carbonum* causes a leaf spot of corn in the Southern states, the spots being small but numerous, targetlike, with buff centers and reddish-brown margins. The lesions become covered with conidia, and ears are attacked with a smutty decay. Both diseases are spread from plant to plant by these spores in the fashion of rusts, and are favored by low, wet locations and rainy seasons. Some of the present-day hybrid corns have resistance to *Helminthosporium* leaf spot, and taking advantage of this resistance will probably be the solution of the problem.

Brown spot of rice, caused by *Helminthosporium oryzae*, is one of the most serious diseases of this crop in the United States. Seedlings may be killed, leaves are affected with reddish-brown, large or small spots, the stalk at the base of the head may be rotted through ("rotten neck"), and yields are notably reduced. The hulls and kernels may be discolored, producing "pecky rice" which is low in milling quality as well as appearance. Control is chiefly through the use of resistant varieties.

In 1944 a new *Helminthosporium* disease of oats was first observed in Iowa. By 1946 it had become widespread in most oat-growing sections of the United States, and that year it caused a loss of one-fourth of the Iowa oats crop. The causal fungus has been named *H. victoriae*, and the disease Victoria blight, since it primarily attacks the newer rust- and smut-resistant oats varieties having the Victoria variety as a parent or grandparent. Susceptible varieties that are menaced by the disease were being grown on 25,000,000 acres in 1946, and include such important varieties as Vicland, Tama, Boone, Cedar, Osage, Neosho, Forvic, Fultex, Victoria, and Ventura. In general the older standard varieties are resistant, as are the rust-resistant derivatives of Bond, particularly Clinton and Benton.

Victoria blight destroys seedlings, dwarfs older plants, rotting their

roots and often killing them before maturity, and decays the stems, causing them to break over. The fungus is both seed- and soil-borne, and the seed-borne infestation can be controlled by seed dusting with Ceresan. In addition growers are warned not to plant susceptible oats varieties on land that has previously grown a diseased crop, and to give preference to the blight-resistant varieties. This disease is a serious blow to the oats breeding program, and will necessitate replacement of many of the newer varieties with others, bred for combined resistance to rusts, smuts, and Victoria blight. It illustrates the potential dangers of "minor diseases" and the difficulties that beset the work of plant breeder and plant pathologist.

EARLY BLIGHT OF POTATO AND TOMATO (*Alternaria solani*)

Alternaria is a genus of sooty molds closely related to *Helminthosporium*, but with dark, pear- or club-shaped conidia divided lengthwise and crosswise into several cells. These are borne over the surface of the mycelium or on conidiophores that emerge from the host tissues in the fashion of *Helminthosporium*. (See Fig. 129.)

Alternaria species are among the commonest saprophytes on decaying vegetation of all kinds. The familiar brownish-gray color of corn stalks in winter, or of stubble fields, is often due largely to the overgrowth of the dead tissues with mycelium and conidia of saprophytic *Alternaria*. A few *Alternaria* species, however, can attack living plants. One of these produces boll rot of cotton, which will be considered in a later chapter, and another, *Alternaria solani*, causes the rather important early blight disease of potato, tomato, eggplant, and other members of the potato family.

On either potato or tomato, early blight takes the form of brown leaf spots marked with concentric rings to give a target effect, which slowly enlarge and may eventually destroy the leaves (Fig. 91). This reduces the yield and exposes tomato fruits to sunburn. In tomato the fungus also causes a stem canker or collar rot of young seedlings, sunken spots or cankers on older stems, blossom drop and loss of young fruits, and dark leathery fruit spots, usually about the point of attachment of the stem. Any of these lesions may show concentric rings, and under moist conditions their surfaces are darkened with conidiophores and conidia. In the case of potato, circular, decayed lesions may form on the tubers, permitting the entrance of decay organisms, and in severe attacks lesions form on the upper stems and petioles.

All common commercial varieties of potatoes and tomatoes are susceptible to early blight. Resistance exists in some breeding stocks and use is being made of these in efforts to incorporate resistance into suitable commercial varieties. The fungus survives in the soil, and seed or tuber

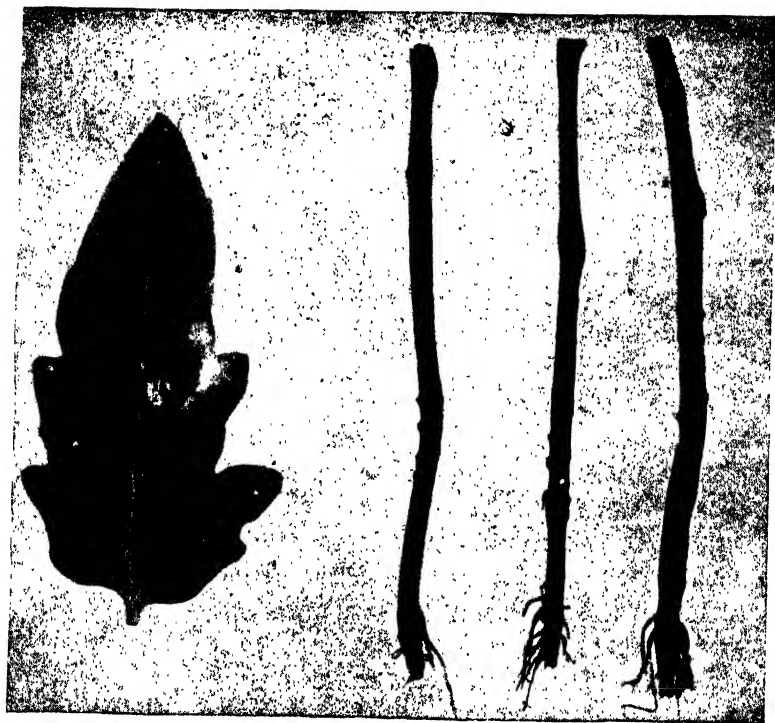


FIG. 91. Early blight of tomato. (*Left*) Targetlike leaf spot. (*Right*) Affected stems, the stage of the disease often called collar rot. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

transmission is of no great importance in producing primary infections, for which reason seed selection or treatment has little value in early blight control. Here principal reliance is placed on a spray program.

For potatoes the most suitable spray materials for controlling early blight, improving yields, and avoiding spray injury are zinc dimethyl dithiocarbamate (Methasan, Zerlate, Zimate, etc.) as a spray, 2 lbs. per 100 gal. of water or as a dust, 10 lbs. in 90 lbs. of talc; Bordeaux mixture, 5-5-50; or Dithane with zinc sulfate and lime. The fixed copper sprays and dusts have given less favorable results. The fungicide is applied every 10 to 14 days after the plants are 6 in. high.

For tomatoes, which are more subject to spray injury, Dithane plus zinc sulfate and lime, Tersan, Bordeaux 4-4-50, and zinc dimethyl dithiocarbamate have given good results in various states, usually better than with the fixed coppers. Fermate is not very effective against early blight but gives excellent control of another important tomato disease, anthracnose. When both diseases attack, good protection has been obtained by alternating applications of one of the fungicides recommended

for early blight control with applications of Fermate. The tomato applications are put on at 10- to 14-day intervals beginning when the plants fall over in the field. Seedbed sanitation, the use of noninfested or sterilized soil for tomato plant propagation, spraying of the seedbed with weak (2-2-50) Bordeaux mixture, use of certified tomato plants, rejection of plants from blight-infested beds, and rapid, careful harvesting, packing, and marketing of tomato plants, are all practices that contribute to early blight control.

Spraying or dusting tomatoes to control defoliation diseases is not always to be recommended. Early-maturing crops often escape leaf blight



FIG. 92. Tomato leaf mold. Leaves showing large chlorotic lesions darkened in the centers by masses of mycelium and conidia.

damage, and spraying such crops is not economical. Spraying sometimes delays maturity, with loss of the advantage of early season high prices. Spraying and dusting are an advantage chiefly to the grower who sells in a midseason to late, quality market.

TOMATO LEAF MOLD (*Cladosporium fulvum*)

Also among the sooty molds are species of *Cladosporium*, with 1- or 2-celled brown conidia borne on short brown conidiophores. Some of these are common saprophytes; a few are pathogens, including the fungi that cause peach scab and tomato leaf mold.

Leaf mold or mildew is a leading disease of greenhouse tomatoes, and also occurs in the field, especially near greenhouses used for tomato culture. Affected leaves show large pale or yellow spots which soon become covered on the under side with a brownish mold consisting of many conidia (Fig. 92). The leaf tissues are killed, the leaves die, and as a result production may be curtailed.

The conidia are spread by splashing water, insects, and air currents, and require high humidity for germination. Some control is obtained through maximum ventilation of the greenhouse and avoidance of sharp drops in greenhouse temperature at night, which raise the relative humidity. Control by the use of fungicidal sprays or dusts is limited by the difficulty in keeping an adequate coating of the fungicide on the lower surfaces of leaves, but volatilized sulfur is helpful. The best solution lies in the use of tomato varieties that have been bred for leaf mold resistance, particularly Bay State, Vetomold, and Globelle. Discovery of a new race of *C. fulvum* which attacks these varieties as well as important breeding stocks necessitates further breeding efforts and maximal use of greenhouse management as a control measure.



FIG. 93. Scab spots on peach. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

PEACH SCAB

(*Cladosporium carpophilum*)

Scab is one of the common diseases of peach, occurring nearly everywhere that peach crops are grown with the exception of the Pacific states where it is rarely seen. It need not be considered a serious menace, however, since it can be efficiently controlled by spraying. To a lesser extent it is found on plum, nectarine, apricot, and cherry. Scab occurs on twigs

and leaves but is most important on the fruits, where it causes numerous round olive-green to black lesions, sometimes called "freckle" (Fig. 93). In severe cases the lesions run together and the fruit may crack open, often paving the way for brown rot. The fruits may be reduced in size, misshapen, and worthless, but in many cases the injury is mainly or entirely to the appearance of the fruit.

The surface of each lesion is covered with brown jointed conidio-phores each bearing at its tip a brown, oval or slipper-shaped conidium. No perfect stage is known. Spread of the disease during the growing season is by means of wind-blown conidia. Overwintering occurs in tiny ($\frac{1}{8}$ in.) cankers on the twigs, in a mycelial state.

Scab is controlled by cover sprays as for brown rot (see p. 134). Two applications are commonly recommended, the first about three weeks after petal fall, the second three to four weeks before harvest. The same fungicides as used for brown rot are effective against the causal fungus of scab. Pruning and orchard sanitation are not essential parts of peach scab control.

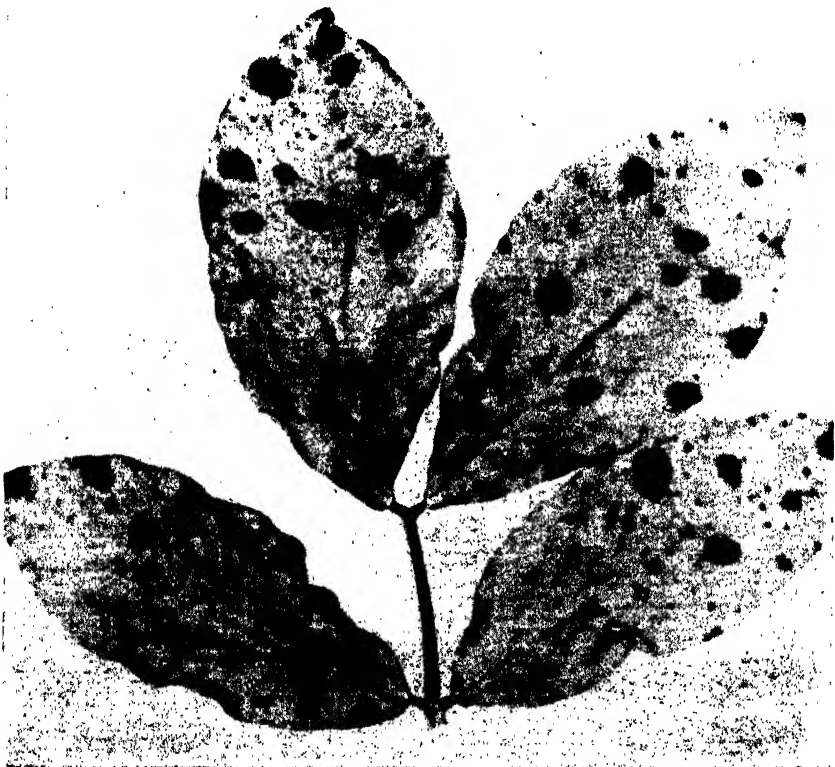


FIG. 94. *Cercospora* leaf spot of peanut.

CERCOSPORA DISEASES; GRAY MOLDS

Cercospora, a genus of the sooty molds with long, wormlike or threadlike, pale to dark conidia often borne in clusters, includes a great many species causing leaf spot diseases of numerous cultivated and wild plants. The *Cercospora* leaf spot of peanuts (*C. personata*) is the leading disease of



FIG. 95. Common *Cercospora* leaf spot of beet.

this crop, and is generally controlled by dusting the fields with sulfur (Fig. 94). This widespread practice and the high margin of profit in favor of dusting suggests that similar returns might be realized from sulfur dusting of other field legumes that regularly suffer from similar defoliation diseases. A *Cercospora* leaf spot (*C. oryzae*) is one of the leading rice diseases. It has been controlled readily by the use of numerous resistant commercial rice varieties, but by 1946 all of these had fallen victim to new races of the fungus.



FIG. 96. Gray mold or mycelial neck rot of onions. (Courtesy, J. C. Walker, Wis. Agr. Exp. Sta.)

The very common leaf spot disease of garden, forage, and sugar beets is caused by *Cercospora beticola* (Fig. 95). It can be controlled by wide spacing of the beets and applications of an insoluble copper dust, although the use of fungicides is not always economically justifiable. Celery is attacked by a *Cercospora* (*C. apii*), controlled by sanitation and frequent spraying or dusting as for celery late blight (see p. 208).

Cercospora leaf spots are very common on trees and ornamental shrubs and, where they are damaging or objectionable, they can be controlled by spraying or dusting.

The genus *Botrytis* includes a large number of species of fungi causing "gray mold" of many fruits, vegetables, and ornamental plants (Fig. 96). Conidia are formed in clusters, like bunches of grapes, on conidiophores that are often so numerous as to cause a dense moldy overgrowth of affected tissues. Some of the gray mold species form conspicuous sclerotia that serve as resting bodies to perpetuate the fungi, and in a number of cases *Botrytis* species are imperfect stages of ascomycetes related to the fungus causing brown rot of stone fruits. Low temperature storage of fruits and vegetables, garden sanitation, and avoidance of excessive humidity about plants are principal measures directed against the gray molds.

Anthracnose Diseases Caused by Melanconiales

The *Melanconiales* is a large order of the *Fungi imperfecti* containing many important parasitic species. The mycelium grows within the host

tissue. The fruiting mass, consisting of a flattish or shallow cuplike basal layer of fungus tissue—the stroma—with the conidia, often borne on short, narrow conidiophores, is called an *acervulus*, meaning a little heap. The expansion of the acervulus ruptures the cuticle or epidermis, permitting the escape of the spores. (See Figs. 37 and 100.) The common term *anthracnose* is applied to any disease caused by a member of this order.

The two leading genera of anthracnose fungi are *Colletotrichum* and *Gloeosporium*, both with clear, one-celled, round or oblong conidia. In *Colletotrichum* the acervuli are distinguished by the presence of long, dark bristles (*setae*) among the conidia; these are lacking in the otherwise similar *Gloeosporium*. (See Fig. 100.) For some of the anthracnose fungi, ascospore stages have been found, and two such cases have been discussed among diseases caused by *Ascomycetes* (cane fruit anthracnose; red rot of sugar cane). The cotton anthracnose is considered later as a part of the cotton seedling disease and boll rot complex (Chapter 9).

CEREAL ANTHRACNOSE (*Colletotrichum graminicolum*)

Distribution and Importance. *Colletotrichum graminicolum* has been observed in western Europe: Germany, Italy, and France, in Canada, and in the United States from southern New England south to Florida and west to the 100th meridian including Oklahoma, the eastern half of Texas, most of Kansas, up to eastern South Dakota.

The statement is frequently encountered that cereal anthracnose is a disease of minor importance and in dry seasons and locations this is doubtless true. The disease lesions are less conspicuous than those of rusts, smuts, and other cereal diseases. Nevertheless, the outbreaks recorded in the *Plant Disease Reporter* indicate that under favorable conditions, especially in the central and southern cereal areas, it may be very destructive. Fields of wheat and rye have frequently been found with 100 per cent of the plants affected and showing losses due to anthracnose from a trace up to total failure as in Kentucky in 1932. In 1918 the disease was epiphytotic; the losses in individual rye fields were as high as 33 per cent in Minnesota, 50 per cent in Missouri and 50 to 60 per cent in Oklahoma. The same year the wheat in Virginia suffered a 10 per cent loss from anthracnose and the Ohio wheat crop 4 per cent with 2 to 26 per cent infestation in every county inspected. Again in 1923 there was a severe outbreak with several states reporting anthracnose as the worst rye disease of the year, the losses reaching 10 per cent in Mississippi and 15 per cent in Ohio. Ten per cent losses were suffered again in 1924 in the southern rye areas. By 1926 Ohio had come to the conclusion that over the preceding nine years anthracnose had been one of the most destructive diseases in the state, and

in Pennsylvania it outranked any other two rye diseases put together. Serious losses in oats were seen in Mississippi in 1926 and in Arkansas in 1931. Missouri in 1928 found anthracnose severe on Sudan grass with a 5 to 10 per cent loss for the state. Losses are due both to lowered yield and poorer quality of affected grain crops.

In 1941 and to a greater extent in 1942 there was a most destructive development of what appeared to be a new stalk rot of broom corn in Illinois with a drop in production from 9,100 tons in 1940 to 2,900 tons in 1942, nearly a million dollar loss for which this disease was largely responsible, and many fields had total losses. This was shown to be caused by *C. graminicolum*, possibly a new strain of the anthracnose fungus which had long been known as causing a leaf disease of broom corn and other sorghums.

Host Plants. The fungus *Colletotrichum graminicolum* has a wide host range among the *Gramineae*. Of the cultivated hosts, rye is most important, followed closely by wheat, oats, and sorghums, including Johnson grass and Sudan grass. Corn and barley are less severely attacked. Numerous grasses are affected and the disease has been reported also on clover, but apparently is unimportant on this host.

Varieties of cultivated cereals differ in susceptibility to anthracnose but lists of resistant varieties are not available. In wheat, Turkey, Kanred, Fultz, Harvest Queen, and Marquis have all been found highly susceptible. Some of the oat hybrids of recent origin have been observed to be free of the disease in Arkansas even though adjacent to other, heavily infested varieties.

Symptoms and Signs. In wheat and rye the disease is distributed at random among plants in the field; in oats where root decay is a prominent feature of the disease, the affected plants may occur in well-defined spots. Infection is most commonly observed at the foot or base of the stem, especially just below the nodes and on the leaf sheaths. The affected tissues are dark and speckled with numerous black acervuli (Fig. 97). The heads may be attacked directly, much in the fashion of scab. The spikelets are darkened, the glumes are dotted with dark acervuli, and the grain within may be shriveled and dark, or in rye the entire head above the point of attack may be bleached and blighted. Premature ripening often occurs. A certain amount of root decay accompanies these above-ground symptoms in oats, especially in the seminal roots, roots formed later escaping injury to a greater degree. *Colletotrichum graminicolum* also attacks the leaves directly, producing spots that vary in appearance with the host species. On corn they are oval or elliptical, brown, studded with acervuli, while on sorghum they are oval to spindle-shaped and brilliant

red or stained brown or of the same color as the light brown dead leaf, depending on the variety of sorghum. As a consequence of stem, leaf, and root infections, plants are stunted in size, the grain is poorly filled, and the general picture, resembling that of a leaf rust attack, is one of malnutrition or starvation. In broom corn the disease is a combination of leaf



FIG. 97. Anthracnose on wheat. Discoloration of stem joints is a characteristic of the disease, as is also the presence of black specks on stems, sheaths, and glumes. Severely infected plants ripen prematurely and bear shriveled grain. (Courtesy, Illinois Natural History Survey.)

spotting, root rot, and stalk rot. Stalk infection involves the entire stalk from base to brush, with red internal discoloration, hollowing of the lower part of the stalk, and lodging.

Etiology. *C. graminicolum* has no known sexual or perfect stage, although in related species of *Colletotrichum* the perfect stages, where known, fall into the ascomycetous genera *Glomerella* (e.g., the cotton anthracnose fungus to be considered later) and *Pseudopeziza* (kin to the alfalfa leaf spot fungus). The only spores produced are conidia, which are spindle- or boat-shaped, about six times as long as broad, clear, with two or more oil droplets, borne on very short conidiophores. The conidiophores and conidia are grouped in circular or oval acervuli up to 1 mm. in diameter, arising from a compact layer of fungus tissue. These are dark owing to the presence of numerous brown spinelike setae, visible as black hairs under low-power magnification.

C. graminicolum overwinters as a saprophytic mycelium or in the form of resting, sclerotiumlike bodies in the soil or in stubble and straw from

previous crops, as spores on the surface of seed grain, or as active, parasitic mycelium in winter grains. In the spring, conidia are liberated from overwintered lesions, are spread by wind and rain to healthy plants, and induce primary infection. In the lesions thus formed, groups of conidia-bearing acervuli soon form, the spores serving for secondary spread, first from stem to stem, later to the heads when these appear.

In oats, a distinct type of life history is seen. Here the fungus is soil-borne. The first roots formed by the germinating seed are highly susceptible; these are rapidly invaded by the soil-borne mycelium and their tissues broken down. The vessels of these roots are plugged with mycelium, and the poor functioning of the roots produces stunting and suffering in the seedling. On the rhizome and base of the stem are formed dark, compact sclerotiumlike fungus masses (see illustrations in *Sci. Agr.* **15**, 370–376 (1935)). These do not produce spores unless above ground. The roots formed subsequently appear to be more resistant to the fungus and escape serious injury, but in the culm the disease progresses in typical fashion.

Epiphytology. There is little exact information on the relation of environment to cereal anthracnose. The disease is favored by warm temperatures but tolerant of a wide temperature range as indicated by its distribution from Canada to Florida and Texas with greatest losses from Tennessee southward. Moisture plainly is an important factor, restricting the disease to areas east of the 100th meridian, and to moist locations and years. Anthracnose is so commonly seen coexistent with cereal rusts that it appears that a similar sequence of rainy or dewy weather favors both types of disease.

Little is known of the relation of soil conditions to anthracnose. Dickson suggests that attention to adequate soil fertility is useful in anthracnose control, but this may merely serve as partial compensation for devitalizing effects of the disease. Coexistence with rusts implies that well-fertilized plants are susceptible to anthracnose. A number of workers indicate that the effects of anthracnose are most pronounced in combination with malnutrition and simultaneous attacks by other diseases.

Control. Because of the many points in connection with anthracnose that lack sufficient study, recommendations on control are theoretical, based on available information on the life history and not well supported by critical tests. These recommendations include:

1. **SEED TREATMENT.** Formaldehyde treatment is suggested to kill surface-borne spores and probably may be attributed to dependence on early data (1909) before organic dusts and copper carbonate came into general use. It is probable that any of the surface treatments used for smut control would be effective in destroying infestation on seed surfaces. Fanning seed to remove light kernels is recommended also.

2. **SANITATION AND ROTATION.** Considering the wide host range of *C. graminicolum* among the cereals and lack of knowledge on physiologic specialization, it is advisable to alternate cereals with noncereal crops wherever practicable. One of the most destructive outbreaks of anthrac-

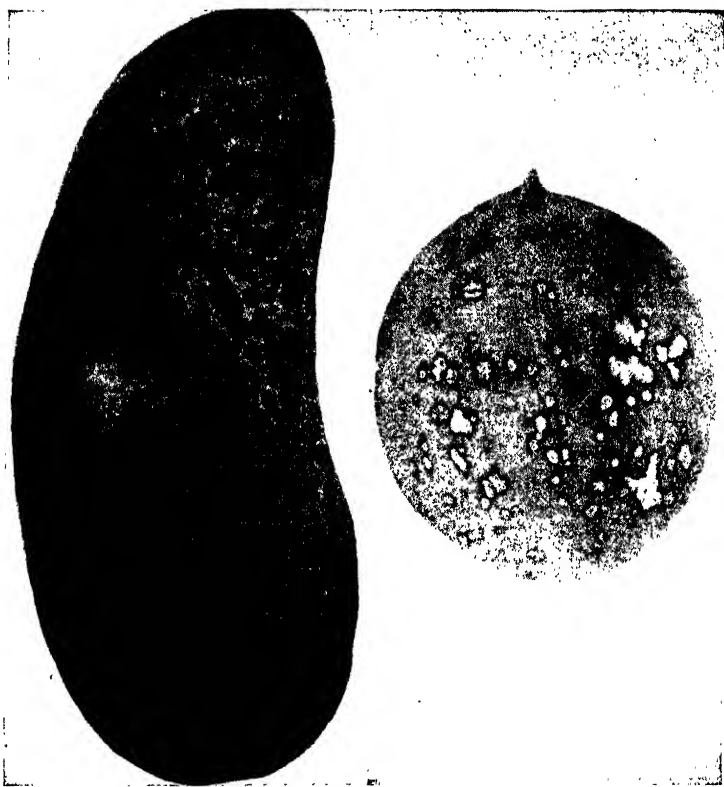


FIG. 98. Cucurbit anthracnose on watermelon (advanced stage) and honey dew melon. (Courtesy, C. J. Nusbaum, S. C. Agr. Exp. Sta.)

nose on record, resulting in complete killing of winter wheat in Kentucky, was in wheat planted on disked corn stubble, although corn and barley are less subject to the disease than wheat, oats, and rye. Thorough turning under of cereal debris, allowing it to decompose well before replanting cereal crops, would doubtless effect important reduction of inoculum.

3. RESISTANT VARIETIES. Hope for the future in anthracnose control rests largely on the varietal differences in susceptibility that have been noted frequently. No specific recommendations on resistant varieties can be made at present, but growers should be alert to detect differences in susceptibility of the major cereal crop varieties, and should be governed accordingly. There is need for a careful study of the resistance of cereal varieties to this disease.

MELON ANTHRACNOSE (*Colletotrichum lagenarium*)

This disease is mainly important on watermelons and muskmelons where it ranks as a major disease; it is sometimes quite damaging to cu-

cumbers but pumpkins, squashes, and gourds are less commonly affected. Its destructiveness is illustrated by an outbreak involving one-fourth of the Oklahoma watermelon crop in 1944; at some shipping points 75 per cent of the melons were unmarketable, and the disease cost Oklahoma growers \$80,000 that year. One-fourth of the Iowa watermelon crop of 1943 was ruined by anthracnose. The vines may be completely destroyed before the fruits mature, the fruits themselves may be ruined in the field, or the principal loss may be from poor appearance and decay in



FIG. 99. Bean anthracnose. Typical pod lesions.

transit. The disease prevails throughout the melon areas east of the Rocky Mountains. The symptoms include irregular, brown or black dry leaf spots, eventually causing the leaf to shrivel up and die and, on the fruit, sunken, circular to irregular rotten areas from pinpoint size to very extensive patches (Fig. 98). Elongate stem and petiole lesions occur. Under moist conditions the fruit lesions are dotted with masses of pink conidia. These rapidly spread the infection in moist weather. The fungus overwinters in decaying vines and on seed from diseased fruits. Temperatures above 63°F. are favorable with optimal spore germination at 72° to 80°F.

Control depends on a three-year crop rotation with noncucurbits, seed treatment with mercuric chloride, 1:1000 for 5 to 10 minutes followed

by water rinse, and spraying or dusting the vines. Bordeaux mixture spray, 4-4-50, is effective against anthracnose but likely to injure or stunt the vines. Good results without injury have been obtained with insoluble copper sprays or dusts (20-80) and with Fermate dust. Usually three or four applications are recommended, at 10- to 14-day intervals after the vines begin to run. Most commercial watermelons are highly susceptible, but the Iowa hybrids, Black Kleckley, Kleckley Hybrid, and Dixie Hybrid, and the Georgia home garden melon, No. 2, are resistant.

ANTHRACNOSE OF BEANS (*Colletotrichum lindemuthianum*)

This major bean disease, often incorrectly called "rust," is of world-wide distribution and occurs in every state in the Union though it is most destructive in the North Central and Eastern states. Anthracnose is noticeable in the pods as sunken, irregular, brown lesions exuding flesh-colored spore masses when moist (Figs. 99, 100). Leaf lesions are on the under sides, along the veins, narrow, and dark red to black in color. Dark, sunken cankers occur on stems and petioles. The dry beans show dark discolored spots that are most obvious on white-coated beans (Fig. 101). These lesions may involve as much as half the seed surface, and may reach downward into the cotyledons. The fungus overwinters primarily in such seed but it may also survive for two years in the soil. Spores produced on the lesions are spread mainly by splashing rain, infection being favored by cool temperatures (72° to 73°F. is optimum for growth of the fungus and none occurs above 86° to 93°F.). The fungus exhibits physiologic specialization, 34 strains in 3 groups having been recognized. In spite of this there has been some success in selecting and breeding beans for anthracnose resistance, the varieties with more notable resistance including Perry Marrow, Genessee, Wells Red Kidney, White Imperial, Idaho Refugee, Wisconsin Refugee, Corbett Refugee, Sugar Pearl and pea bean No. 22.

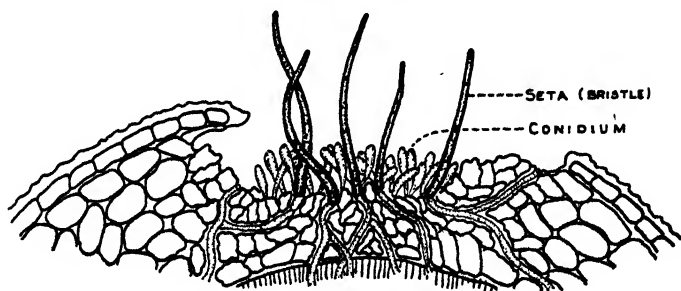


FIG. 100. Acervulus of the bean anthracnose fungus. The ruptured host tissues are a constant feature of anthracnose lesions, and the presence of the setae distinguishes anthracnose fungi of the genus *Colletotrichum* from other closely related organisms. (After Whetzel and Schwarze.)

Control. Control depends first on the use of anthracnose-free seed. This can be obtained from areas in which anthracnose does not become prevalent, notably Colorado, Idaho, and California. For the best results, this should be assisted by a rotation, using beans on the infested land only after two years in another crop, and avoiding picking or handling the vines when wet, a precaution that is paramount also in controlling bacterial blight of beans. Seed treatments are of little or no value in controlling this disease. Other factors being equal, preference should be given to the anthracnose-resistant varieties in areas of common infestation. Ordinarily spraying or dusting is not used in controlling anthracnose, but under conditions of heavy attack spraying with Fermate, Puratized, Dithane, Phygon, or Bordeaux mixture has been beneficial. In New York tests five applications of Fermate at 2 lbs. per 100 gal. saved about 30 per cent of the pods from anthracnose.

OTHER ANTHRACNOSE DISEASES

Cotton anthracnose (*Glomerella gossypii*) is considered in Chapter 9.

Flax anthracnose (*Colletotrichum lini*) ordinarily is not a very serious disease but is as capable as wilt of destroying plants. It is outstanding in being internally seed-borne and is controlled by the use of bright, plump, clean seed from disease-free crops.

Onion smudge (*Colletotrichum circinans*), while not a major onion disease, is distinguished as one of the few cases for which we have a clear-cut picture of the nature of resistance ascribed, in red and yellow onions, to the pigments protocatechuic acid and catechol (see p. 446).

Southern clover-alfalfa anthracnose (*Colletotrichum trifolii*) has been quite destructive in the red clover area from Missouri to North Carolina. It is controlled in red clover by use of the anthracnose-resistant strain developed in Tennessee (Fig. 102) or the variety Cumberland.

Tomato anthracnose (*Colletotrichum phomoides*) commonly affects tomato fruits with round, sunken spots that are first watersoaked, later dark, depressed, with targetlike rings, covered with masses of salmon-colored



FIG. 101. Bean anthracnose on the seed. The seed is infected through the cankers on the pods. Infected seed serves as a means of infesting the new crop and should never be planted. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

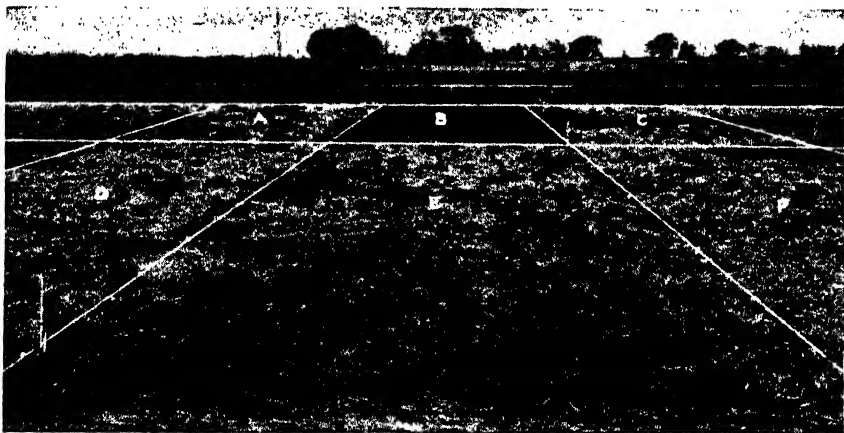


FIG. 102. Destruction by anthracnose of red clover of several strains (A, C, D, E, and F) (from Italy, Netherlands, Poland, Oregon, and Denmark, respectively), while the resistant Tennessee strain (B) was uninjured. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

spores in moist weather. The disease is a serious factor in tomato processing. It is controlled by crop rotation and spraying or dusting with a copper fungicide, zinc dimethyl dithiocarbamate (Zerlate, etc.) or Fermate.

Pestalotia species, distinguished by conidia equipped with hairlike appendages, are common on many kinds of woody plants, but generally are considered weak parasites or saprophytes.

Marssonina populi, with two-celled conidia, causes the very common leaf spot disease of poplar and cottonwood.

Many other anthracnose fungi, particularly species of *Colletotrichum* and *Gloeosporium* might be mentioned. In fact, there is hardly a major crop that does not suffer at one time or another from pathogens belonging to this group.

Diseases Caused by Sphaeropsidales

The *Sphaeropsidales* are those imperfect fungi in which the conidia are produced in dark, flasklike pycnidia from which the spores often ooze in long tendrils. (Fig. 37.)

APPLE BLOTCH (*Phyllosticta solitaria*)

The word "*Phyllosticta*" means "leaf-spot," and there are some 800 species of the imperfect fungus genus *Phyllosticta*, usually associated with leaf spot diseases of many species of plants. The genus *Phoma*, containing 1200 species, is indistinguishable from *Phyllosticta* except that usually it occurs on woody or fleshy tissues rather than on leaves. Both genera are

characterized by clear, one-celled conidia, produced in simple, flasklike, black pycnidia sprinkled over the surface of the spot. Apple blotch represents this type of disease.

History and Distribution. Apple blotch was discovered in Indiana in 1893 and the organism correctly named shortly after. During this early period it was frequently confused with scab and other apple diseases. That the disease was important from the outset is indicated by attempts at control by spraying and dusting in Illinois, 1903, and Arkansas, 1906. In 1907, two Arkansas workers, Scott and Rorer, showed by infection experiments that the leaf spot, fruit blotch, and bark canker are all stages of the same disease. They also investigated the etiology and control of the disease and the mycology and life history of the causal fungus. Since their time most of the work on the disease has concerned control by spraying. The most extensive monograph on apple blotch is the paper of Guba in 1925 (*Illinois Agr. Exp. Sta., Bull.* 256) to which the reader is referred for a detailed discussion.

Blotch occurs in a rectangular area extending from Pennsylvania to Nebraska and south to include all southern states except Florida. It appears to be a native disease of the American crab apple.

Importance. Blotch is most destructive in the Ozark section of Missouri, Arkansas, Oklahoma, and in Texas, where it frequently outranks scab and becomes the leading apple disease. Losses up to 10 per cent annually are reported by individual states, and the losses in the national crop range from 500,000 bushels to more than 4,000,000 bushels annually. The loss is due to the following factors: lowered market value of the fruit because of disfiguring blemishes, predisposition to fruit decay, defoliation with ensuing weakening of the tree, to a lesser extent injury to the tree from bark cankers, rejection of affected nursery stock, and cost of control measures.

Host Plants. *Phyllosticta solitaria* is limited in its attack to apples and crab apples. Most commercial apple varieties are very susceptible to blotch but some varieties are quite resistant unless exposed to very heavy inoculum. The more resistant varieties include Grimes Golden, Jonathan, Stayman Winesap, and Winesap.

Symptoms and Signs. Blotch symptoms occur on leaves, fruit, and bark (Fig. 103).

Leafspots due to the blotch fungus are small, about 1 mm. in diameter, although often very numerous on the leaf, round, white, usually with a tiny, solitary black pycnidium in the center of each spot, occasioning the name *Phyllosticta solitaria*. They vary somewhat in size, depending on the apple variety. Larger, elongate lesions occur along the veins and midrib.



FIG. 103. Apple blotch. (*Left*) Cankers on apple twig, enlarged, showing the tiny black pycnidia, spores from which initiate spring infections. (*Right, top*) Typical blotch leaf lesions, at the center of each of which is a tiny black pycnidium, not visible in the photograph. (*Right, bottom*) Typical superficial fruit lesions.

The leaf areas between the spots usually are normal green, but where the spots are very numerous the leaf falls prematurely.

Fruit blotch occurs as brown patches very irregular in outline, sometimes star-shaped, feathery at the margin. These lesions are studded with numerous tiny, black pycnidia. They are superficial, not extending into the apple flesh, but often opening the way for decay organisms that rot the fruit more or less extensively. When the fruit is affected in a very early stage the fruit lesions may develop into deep cracks, a three-angled crack being common.

Bark lesions on the twigs at first are lens-shaped or oval, light in color, smooth, sprinkled with numerous pycnidia. They are biennial; after the second year the wood under the lesion heals and the area becomes rough. Meanwhile, a new lesion area develops around the edges of the old lesion, and this may continue year after year until large rough areas are produced on the branches.

Etiology. *Phyllosticta solitaria* is an imperfect fungus having as its only spore form clear, one-celled, oval conidia borne on slender conidiophores and produced in large quantities within simple, globular to irregularly shaped pycnidia. The formation of pycnidia sometimes is preceded by the heaping together of mycelium to form sterile, dark, overwintering mycelial masses on the lesions, known as pycnosclerotia. The following season these become pycnidia. Ordinarily pycnidia formed early in the season develop spores directly, while those initiated later in the season pass through the pycnosclerotial stage. The spores of *P. solitaria* are unusual in sometimes having a long or short gelatinous appendage that partly envelops the spore (illustrated in Guba's monograph).

Overwintering occurs in the form of pycnosclerotia mainly on bark cankers but to some extent on fallen leaves and fruit, and as dormant mycelium in the bark cankers. Conidia are produced in the spring and these are spread mainly by splashing rain, to a less extent by wind, initiating the new infections on leaves and at the bases of buds or bud scars, later on the fruit. The secondary infections normally occur rather late in the season, several weeks after petal-fall.

Man is the principal agent of dissemination from one orchard to another, infected nursery stock constituting the leading means of introducing the disease into new localities. Neglected orchard or windbreak trees and wild species of *Malus* may serve as reservoirs from which the disease may spread to nearby orchards year after year.

Epiphytology. The pycnidia swell and discharge their spores only when thoroughly wet, following abundant rains. Dews and light rains

of short duration are insufficient for this purpose. Even under favorable moisture conditions pythidia may retain many spores until later in the season. Germination of the spores is dependent on warm temperatures. They germinate promptly at 77° to 86°F. and more slowly at 59° to 68°F. Well-fertilized trees are most susceptible because of their increased succulence, although it is sometimes necessary to fertilize and stimulate trees that have suffered devitalization from blotch, even at the expense of increasing their susceptibility, and to depend on spraying for control of the disease. Juvenile tissues are more susceptible than more mature ones, in fact, fruits become quite resistant to new blotch infections after midseason.

Control: 1. **EXCLUSION.** In starting new orchards, care should be used to secure only blotch-free nursery stock, and to remove any neglected cultivated or wild apple trees from the vicinity of the orchard. The protection given by most state nursery inspection services helps appreciably to minimize danger from infected nursery stock.

2. **SPRAYING.** Spraying is the principal means of blotch control. The cover applications recommended for apple scab (p. 104) are useful in controlling blotch. In the past, Bordeaux mixture 2-4-100 or stronger has been generally used. Fermate, at the rate of 1 to 3 lbs. per 100 gal. has given very good results and is now a standard recommendation for the blotch sprays in some states. Fermate should not be used in combination with Bordeaux or lime or applied before or after Bordeaux applications, because of possible spray injury to fruit and foliage. The advisability of using cover sprays for blotch control, and insecticide applications in addition to, or in combination with, them is a local problem, depending on seasonal, geographical, and local environmental conditions, the cost of spray applications, and the value of the expected crop; it must be worked out for each orchard as an individual matter.

3. **PRUNING.** As an accessory control measure, pruning is useful to produce the type of tree development that is easily and efficiently sprayed. Pruning to remove most or all of the bark lesions is impractical except in the case of lightly infested nursery stock. Surgery to remove larger cankers also is impractical.

4. **FERTILIZATION.** Fertilization and other cultural methods to promote vigor in the trees are often needed in older, blotch-weakened orchards, although this increases susceptibility to blotch.

5. **RESISTANT VARIETIES.** In areas of regular blotch losses, wherever possible varieties for new orchards should be selected from the list of those resistant to blotch.

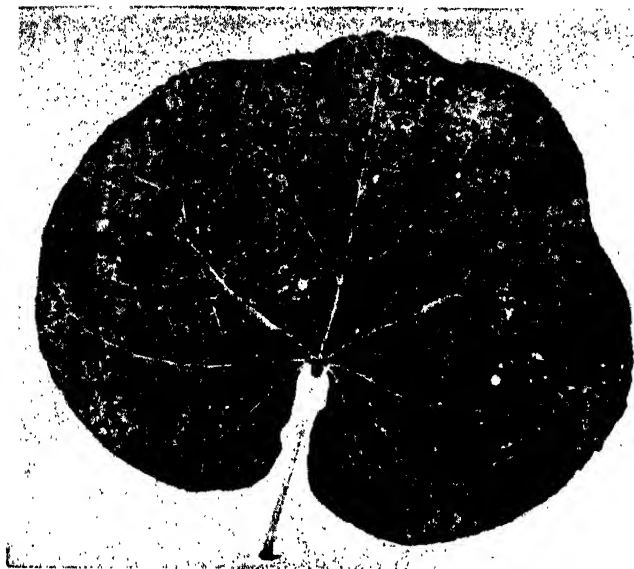


FIG. 104. Representative of the many *Phyllosticta* leaf spots of trees is this one of the redbud.

PHYLLOSTICTA LEAF SPOTS OF OTHER HOSTS

A great many plants suffer from leaf spot diseases due to various species of *Phyllosticta* and other related imperfect fungi (Fig. 104). Often these are quite noticeable on shade and ornamental trees. They are sporadic in occurrence, largely dependent on current moisture conditions for their development. Because of the uncertainty of their appearance in any given year, and the fact that once the spots appear it is too late to control the disease that season, special control methods are rarely recommended. Where the value of such trees is high, they can usually be protected from these leaf spot diseases by repeated applications of Bordeaux mixture or other fungicidal sprays.

ROSE CANKERS (*Coniothyrium* species)

Coniothyrium differs from *Phyllosticta* principally in that the conidia are brown instead of colorless. Two species attack roses, *C. fuckelii*, causing stem canker and graft canker, together called common canker, and *C. wernsdorffiae* which produces brand canker. Two other similar types of disease on roses are brown canker, caused by an ascomycete, *Cryptosporella umbrina*, and crown canker (*Cylindrocladium scoparium*). These four types of canker are distinguished as follows:

Stem Canker; Graft Canker. Pale yellow or reddish spots enlarging and joining to form extensive cankered areas, sometimes girdling the

stem, in which case it dies back to the lesion. Black, fringed leaf lesions, similar to black spot lesions occur (Fig. 105).

Brand Canker. Small, pale brown, oval spots with purple borders enlarging to surround the stem, sharply contrasted against normal green stem tissues between the lesions. Long canes may die back to girdling lesions.

Brown Canker. Small, raised, circular purple spots becoming grayish white and developing into large brown cankers the second season, sometimes surrounded by a reddish border.

Crown Canker. On greenhouse roses only; extensive, black, water-



FIG. 105. Rose cankers. (Top) Typical cases of crown cankers. (Bottom) Common cankers showing the tiny, dark fruiting bodies (pycnidia) bursting through the epidermis. (Courtesy, P. P. Pirone, N. J. Agr. Exp. Sta.)

soaked punky cankered areas at the graft union or just above; plants not killed, but weak with poor blossom production (Fig. 105).

Rose cankers are a sure sign of poorly handled roses. Well nourished, vigorous rose plants have a high degree of resistance to canker, but roses which are weakened by poor culture or marketing methods are readily susceptible to canker. Proper culture and handling constitute the best of all methods for keeping the canker problem to a minimum. Prevention of rose cankers, consequently, may be effected by the following measures:

1. In buying rose bushes, reject any that show discolored or dead areas on the canes. It is preferable to buy plants directly from the nursery, to avoid the weakness of plants that are marketed by those department stores that are not equipped properly to care for plants. Plant rose bushes in their permanent location as promptly as possible.

2. If it becomes necessary to plant cankered rose bushes, all dead parts should be pruned out and burned. Pruning cuts should be made with sharp tools, and branch stubs should not be left. The cut surfaces resulting should be covered with tree paint or shellac. The pruning shears should be disinfested from time to time by dipping in a solution of formaldehyde (1:20) or kerosene or gasoline to which a little lubricating oil has been added.

3. The new growth should be dusted or sprayed at intervals throughout the season, using one of the fungicides recommended for mildew and black spot, the two other common and destructive rose diseases. (See pp. 127 and 138.) The first application is made as soon as the leaves begin to develop, and this is repeated at intervals of two to three weeks throughout the season.

4. No control methods will be satisfactory without adequate attention to the nutrition and watering of the plants. Every effort should be made to insure that the plants are provided with an adequate supply of water and nitrogenous fertilizer, and that they are properly pruned back in the spring.

SEPTORIA DISEASES OF WHEAT

Septoria is an important genus of imperfect fungi characterized by long, threadlike, many-celled conidia produced in black, globose pycnidia. There are over 900 species many of which are of considerable economic importance. Two occur on wheat, *S. nodorum*, cause of glume blotch, and *S. tritici* which produces speckled leaf blotch.

Speckled Leaf Blotch (*Septoria tritici*). This trouble is often confused with winter injury since it results mainly in a killing of leaves during late fall, winter, and early spring. The disease ordinarily disappears by



FIG. 106: Speckled leaf blotch of wheat. Note the many small black pycnidia and the killing resulting from infection. (Enlarged about 3x.)

jointing time. Rye and bluegrass also are attacked. The leaves of affected plants show oval, light brown spots, often with a yellow margin, speckled with minute, dark brown pycnidia which liberate threadlike conidia in large numbers. As the spots increase in number they cut off the water supply of the leaf tips and a progressive dying back of the leaves results (Fig. 106). It is not uncommon to find 50 per cent or more of the leaves killed at the critical time when winter wheat is emerging from its dormant period, resulting in a material retardation in spring development. Occasionally plants may be killed outright. Boewe in Illinois ranks the disease as second in importance on wheat, its damage being exceeded only by leaf

rust. In 1941 the disease was epiphytotic in the winter wheat areas, continuing the destruction of wheat leaves until 40 to 50 per cent of the foliage was destroyed by heading time. This outbreak occurred in a cool, moist spring, and the widespread damage was somewhat obscured by the favorable effect of the abundant rains.



FIG. 107. Glume blotch of wheat. The fungus attacks the nodes most often, discoloring them and producing tiny, black pycnidia. On the glumes small, dark spots are produced. (Courtesy, Illinois Natural History Survey.)

The fungus survives on volunteer wheat, rye, and bluegrass, and in infested debris from the wheat crop. The spores retain viability for a year. Primary infections occur in the cool fall weather and secondary cycles may follow, while with the advent of winter the fungus remains dormant or develops slowly in the wheat leaves. The spores require a very long moist period for germination and infection, and this, rather than temperature relations, seems to limit infection to the cooler seasons, when moisture is more persistent.

Little information is available on control. The following precautionary

measures may be suggested: the use of well cleaned and treated seed, suppression of volunteer grain and grasses between wheat crops, care in threshing to avoid blowing of infected leaf fragments to new fields, plowing soon after harvest where soil conditions permit, to hasten decomposition of infested debris, and crop rotation with at least one year intervening between wheat crops. The wheat varieties Red Chief, Nabob, Prairie, Gladden, Nitrary, Red Rock, and Thorne are resistant to *S. tritici*, and breeding to introduce this resistance into other commercial varieties is progressing in Kansas.

Glume Blotch (*Septoria nodorum*). Glume blotch ordinarily is a disease of minor importance, but may be fairly injurious when there is excessive rainfall between blossoming time and harvest. The fungus also attacks barley, rye, and bluegrass. Various parts of the plant may be attacked but the disease is most conspicuous on the glumes, taking the form of irregular chocolate-brown spots, sprinkled with tiny, black pycnidia which serve to distinguish glume blotch from other diseases attacking the glumes of wheat (Fig. 107). In severe attacks the kernels may become shriveled and the heads reduced to one-half normal size. Leaf spots with brownish margins and light centers dotted with pycnidia sometimes occur, and the fungus also attacks the stems, turning the nodes dark brown and the internodes light brown, resembling late freeze injury.

Septoria nodorum is adapted to high temperatures. The black pycnidia contain countless spores that are carried by the wind from one plant to another. The fungus survives in wheat straw and chaff and as spores on seed surfaces, which may be important sources of primary infections since infested seed often produce infected seedlings. No special control measures are required beyond those already suggested for speckled leaf blotch. Seed treatment with Ceresan, as for bunt, has been shown to prevent seedling attack.

These and other species of *Septoria* often are found associated with leaf spots of pasture grasses.

SEPTORIA LEAF SPOT OF TOMATO (*Septoria lycopersici*)

This is a leading defoliation disease of tomatoes throughout the United States except the Pacific States and the deep South. It destroys the leaves, weakening the plants, interfering with fruit production, and exposing fruits to sunburn.

Leaf spot is recognized as numerous small roundish lesions with dark margins and gray centers, the latter speckled with tiny black pycnidia (Fig. 108). Affected leaves turn yellow, shrivel, and die. The disease becomes most noticeable at the time when fruits are beginning to form,



FIG. 108. *Septoria* leaf spot of tomato. Note the small black pycnidia on the lesions. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

involving the lower leaves and progressing upward until the plant may be almost completely defoliated.

The fungus, *Septoria lycopersici*, also attacks a number of common weeds of the tomato family and it may overwinter on these or in plant residue that has not been plowed deep. Tomatoes in the seedbed may contract the disease, serving to introduce it into field and garden plantings. The long, threadlike conidia which ooze out from the pycnidia during moist weather are spread mainly by splashing rain.

Tomato leaf spot can be greatly reduced by certain cultural practices, including use of disease-free seedlings, from hot-water treated seed grown in regularly sprayed seedbeds, a three-year rotation with new tomato plantings not directly adjacent to those of the year before, and fall eradication of the weed hosts of the fungus, particularly horse nettle. If these practices are not followed, spraying or dusting of the vines may be re-

quired. The insoluble coppers, as sprays or dusts, are often used for this purpose, and good results have been obtained also with Phygon, Dithane with zinc sulfate and lime, Fermate, and Cuprocide. Certain foreign tomato species are resistant to *Septoria* and these are being used in breeding new varieties with leaf spot resistance.

CELERY LATE BLIGHT (*Septoria apii-graveolentis*)

Even in areas where celery commonly is not grown, its most important general disease, late blight, may be seen in the market, where it causes heavy losses, as well as in the field.

The disease takes the form of small or large leaf spots, gray or brown with a darker border, sprinkled with black pycnidia (Fig. 109). Petioles,



FIG. 109. Late blight of celery (*Septoria apii*).

stems, and flower parts are attacked, showing small spots. Under moist conditions the pycnidia exude threadlike conidia that efficiently spread the disease in the field or in storage or transit. The fungus overwinters in crop debris and to some extent in the seed. Control is somewhat aided by disposal of crop refuse, but principal dependence is on spraying or dusting with copper-lime dust, starting in the seedbed and continuing at frequent intervals through the growing season. Approved fungicides for this purpose are certain copper compounds, such as Copper A, copper-lime dust, Bordeaux mixture, Bordeaux plus wettable sulfur, and Cuprocide-sulfur-talc dust. Phygon and Dithane-zinc sulfate-lime are also promising.

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Chapter 8

Diseases Caused by Phycomycetes and Related Fungi

The phycomycetes are the lowest class of fungi, probably evolved from the aquatic algae and ancestral to the ascomycetes and basidiomycetes. They are very diverse in habit, structure, and methods of reproduction, but as a class they show characteristics that readily distinguish them from the higher fungi. Ordinarily the mycelium lacks cross walls except those that set off the sexual organs. The mycelium may be thought of as a complex, branched but single, multinucleate cell. In this nonseptate mycelium, which is often irregular in diameter, the granular and highly refractive cytoplasm can often be seen surging through the hyphae in a powerful and regular streaming. In the lower phycomycetes, nonsexual reproduction usually is by means of free-swimming *zoöspores*, liberated from *zoösporangia*, while in higher forms the zoösporangia may function as wind-blown conidia, germinating either by a germ tube or by the production of zoöspores. (Figs. 110, 117.) In the highest phycomycetes of all, the zoöspore-forming habit has been discarded entirely, and the sporangia produce air-borne, nonmotile *sporangiospores*. (Fig. 111.) Sexual reproduction is through the union of gametes. In the simplest forms two zoöspores may act as gametes, fusing to form a *zygote*, usually a resting body which may germinate later. Higher up the phycomycete scale we find the gametes, male and female being either alike or dissimilar, consisting of special cells, separated by cross walls from the main mycelial system. These unite when growth processes bring them into contact, and a zygote, usually a thick-walled resting spore, is formed. If the two gametes are alike, as in the common bread mold, the resulting fertilized cell is a *zygospor*e (Fig. 111), while if male and female elements are distinct, the fertilized egg cell is an *oöspore* (Fig. 110).

The phycomycetes for the most part are adapted to humid conditions and frequently an important part of their life cycle requires the presence of standing water. Under these conditions we find them associated with destructive plant diseases—the downy mildews of potato, tobacco, spinach, grape, cucumber, and sugar cane, damping-off of seedlings in wet soil,

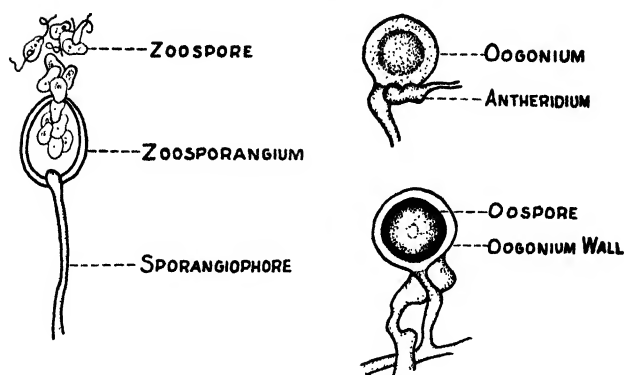


FIG. 110. Reproductive structures of the oömycetes. (Left) Nonsexual reproduction by zoöspore production. (Right) Sexual reproduction with formation of oöspores.

and storage decays in moist environments. Some of them have unusual habits, as the *Entomophthorales*, many of which are parasites on insects and often serve for the natural control of insect epizootics, the *Chytridiales*, primitive fungi often parasitic on algae or other fungi, and the *Saprolegniales*, primarily parasitic on fish and other aquatic animals, sometimes seriously jeopardizing fish populations.

Downy Mildews

LATE BLIGHT (DOWNY MILDEW) OF POTATO AND TOMATO (*Phytophthora infestans*)

History and Distribution. The early history of this classic disease that inaugurated the science of plant pathology was recounted in Chapter 1. Highlights of this history were the introduction of the disease from its ancestral home in America to European potato fields, the disastrous

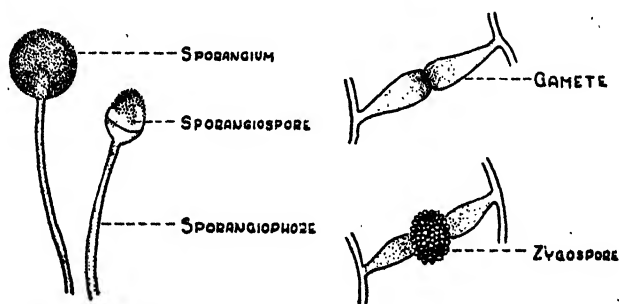


FIG. 111. Reproduction in the zygomycetes, as seen in *Rhizopus nigricans*, the common bread mold. (Left) Nonsexual reproduction by means of sporangiospores. (Right) Sexual reproduction by zygospores.

epiphytotics in the 1840's, resulting in the Irish murrain and famine of 1845-1846, and eventually the discovery of late blight control by the use of Bordeaux sprays (1882-1885). Though severe epiphytotics have occurred since that time they have been less destructive than the epiphytotic of 1845, and with the general use of fungicides today, late blight destructiveness may become largely a thing of the past.

Late blight occurs in nearly every potato-growing area—the United States, Canada, Europe, India, Australia, and New Zealand—and at rather long intervals it breaks out in transient epiphytotics. Today its importance as a disease of potato tubers in storage and of tomatoes in the field compares with its depredations in potato fields.

Importance. Ordinarily, late blight is most destructive in the cooler potato-growing areas, such as the northeastern United States, eastern Canada, and northwestern Europe, and in the deep South where potatoes are grown as a winter crop. Despite very general spraying for late blight control, the losses from this disease often are high. The destructiveness of the disease and its variation from season to season are seen in the following United States loss estimates from the *Plant Disease Reporter* (Table 4).

Table 4
ANNUAL ESTIMATED UNITED STATES LOSSES FROM POTATO LATE
BLIGHT, 1918-1942

<i>Year</i>	<i>Late Blight Loss (Bushels)</i>	<i>Year</i>	<i>Late Blight Loss (Bushels)</i>
1918.....	8,745,000	1929.....	1,753,000
1919.....	20,978,000	1930.....	5,169,000
1920.....	43,257,000	1931.....	4,430,000
1921.....	2,106,000	1932.....	9,230,000
1922.....	11,288,000	1933.....	1,303,000
1923.....	623,000	1934.....	3,409,000
1924.....	21,980,000	1935.....	9,170,000
1925.....	14,278,000	1936.....	3,637,000
1926.....	27,013,000	1937.....	7,141,000
1927.....	26,269,000	1938.....	54,573,000
1928.....	30,998,000	1942.....	25,000,000

These figures show that late blight has lost none of its destructive potentiality. In 1938, nearly a hundred years since the great European epiphytotic and more than 50 years after the discovery of Bordeaux mixture, more than one-eighth of the United States crop was needlessly destroyed by late blight. In that year 45 per cent of the New York crop, 10 per cent of the Maine crop, 35 per cent of the Vermont crop, 15 per cent of the Pennsylvania crop, and 20 per cent of the Virginia crop was sacrificed

to the disease that raged from Maine to Texas. Everywhere the reports agreed that the losses were all exacted from the growers who, indifferent because of the lighter losses of 1929–1937, failed to spray in 1938. (See *Plant Dis. Rep.*, *Supp.* 119, Diseases of plants in the United States in 1938, 1939.) In 1942, 1943, and 1944 there were further destructive outbreaks. The outbreak of 1943 caused a loss of 16 per cent of the Iowa crop, and in Maine, that year, 16 per cent of the stored crop rotted in the bin, leading to further heavy losses in 1944 which was also a severe blight year in the South with 40 per cent of the Louisiana crop lost and several thousand acres destroyed in Mississippi.

The losses consist first in the destruction of the vines, resulting in a short crop, and second in the decay of infected tubers, in the field or in storage. This disease alone does not produce extensive tuber decay, but it is regularly followed by secondary decay organisms that rapidly rot the tubers.

Host Plants. *Phytophthora infestans* affects only plants of the nightshade family, the *Solanaceae*. It is principally important on potato and tomato, in which it is sometimes a major disease, as in 1946, and occurs to a minor extent on petunia and eggplant. It affects various wild *Solanaceae*, sometimes becoming quite destructive on these.

There is considerable variation in susceptibility to late blight among potato varieties and species, however in the past the commercial varieties grown in America have all been quite susceptible. Thanks to intensive breeding the blight-resistant potato varieties Sebago and Sequoia have been developed. These are not immune from blight, but suffer much less than other varieties and permit a greatly reduced spray schedule. Sebago, which compares with Green Mountain, has been called the best late potato for Wisconsin and has performed well in Maine, New York, Florida, Michigan, Minnesota, and western Washington. Recently, however, Sebago has shown signs of losing its resistance to blight, and more highly resistant or immune varieties are needed. Breeders have been aware of this, and new varieties that are resistant or fully immune from blight, such as Empire, Placid, Virgil, Chenango, Ashworth, Potomac, and Menominee, adapted to different regions, recently have been made available to growers.

Symptoms and Signs. The picture of late blight as seen by the layman is brought out in the following description in a letter from Canada to Dr. Bellingham of Dublin, 1844.

Toward the close of the month of August I observed the leaves to be marked with black spots, as if ink has been sprinkled over them. They began to wither, emitting an offensive odor; and before a fortnight the field, which

had been singularly luxuriant and almost rank, became arid and dried up, as if by a severe frost. I had the potatoes dry out during the month of September, when about two-thirds were either positively rotten, partly decayed and swarming with worms, or spotted with brownish colored patches resembling flesh that had been frost-bitten. These parts were soft to the touch and upon the decayed potatoes I observed a whitish substance like mold.

The disease often occurs in rapidly expanding circular areas in the field. On individual plants the necrosis passes upward from the lower leaves, and petioles and stems as well as leaves become black and dry (Fig. 112). The tuber lesions resemble shallow pits which deepen when secondary decay sets in. The frostlike mildew, occurring especially on the lower surfaces of leaves at the margins of the lesions, consists of multitudes of white, branched sporangiophores, tipped with ovoid sporeangia.

On tomatoes a similar rapid necrosis kills the leaves and eventually the vines. Stem lesions are dull-brown cankers that may split open. The fruits are attacked when green, with large, poorly defined, brownish lesions, involving up to half the fruit or more. Affected areas do not ripen, and thus contrast sharply with the remaining part of the fruit. The rot is firm, ordinarily not deep seated. Sporangiophores appear on the foliage



FIG. 112. Late blight of potato. (A) Underside of blighted potato leaf showing watersoaked spots from which the cobwebby growth of the fungus develops. (B) Sunken areas on the tubers indicate early stages of late blight rot. (C) Penetration of late blight rot into the flesh of the tuber. (Courtesy, J. H. Muncie, Mich. Agr. Exp. Sta., from negatives made by G. H. Coons.)

lesions but less commonly on the fruits unless these are growing under very moist conditions. Tomatoes may be attacked in the seedbed and in this case the affected plants are means for spread of the disease to new localities.

Etiology. The fungus overwinters as mycelium in affected tubers, and perhaps also in the soil as mycelium or oöspores. The primary infections ordinarily develop from infected shoots produced by diseased seed tubers or waste tubers in dump heaps. On the surface of these first infected sprouts a moldy coating of sporangiophores appears. In cool weather (54°F.), within the egg-shaped zoösporangia are produced a quantity of free-swimming, biciliate zoöspores. These are distributed by splashing rain. In northern areas the first infections may be traceable to blight-infected southern-grown tomato plants. On coming to rest in a suitable infection court, the zoöspore or swarm spore resorbs its cilia and protrudes a germ tube or infection hypha that grows through a stoma and on coming in contact with cells of the mesophyll commences its parasitic feeding. This is intracellular, the mycelium penetrating the cells and ingesting their substance through long, coiled, threadlike haustoria. Further development of the mycelium occurs and more and more cells are invaded and destroyed until in about five days necrosis is obvious to the unaided eye.

Under warmer conditions at 75°F. no zoöspores are formed; instead, the sporangium as a whole breaks off and is blown or washed about, ultimately germinating by an infection thread.

The infected foliage soon produces a crop of secondary sporangia, and the process of infection is repeated rapidly until large areas of the field are destroyed.

Some of the zoöspores are washed down the stems into the soil and in contact with the developing tubers, where they germinate and initiate the tuber lesions that result in overwintering of the fungus. The tubers may become contaminated also by contact with the vines if dug when the vines are still green. There is evidence to indicate that the fungus may develop to some extent as a saprophyte in the soil on the decaying remains of the crop, and perhaps even overwinter in that condition, but this is not of sufficient practical importance to require consideration in the control program.

The perfect stage, oöspore production, occurs rarely in *Phytophthora infestans*, in contrast with most other downy mildews, and seems to play little part in the life history of the pathogen. It occurs in culture but has not been found in the potato plant itself. According to Clinton, who first thoroughly studied them in 1909–1910, the oögonia or female organs appear as swollen thick-walled hyphal tips, single or double. Within the

oögonium is the egg or oösphere. The male organs, *antheridia*, appear even more rarely than the oögonia. These are thin-walled, club-shaped protrusions of the mycelium that come into contact with the oögonium, and fertilize it by the passage of nuclei from antheridium to oösphere. Following this, the fertilized oösphere becomes thick-walled and acts as a resting spore.

Prior to 1876 De Bary proved the causal relationship of *P. infestans* to blight, fulfilling the requirements of Koch's rules of proof (see p. 399). The fungus evidently exists in more than one physiologic form, the fungi on potato and tomato each being particularly adapted to its host.

Epiphytology. Reference to the figures on annual losses shows clearly that late blight is a disease that fluctuates in severity from year to year, depending on the seasonal weather. The most important conditions favoring epiphytotic development are continued humid weather and cool temperatures, 60°F. or lower, especially at night. Germination of the sporangia is most active at 50° to 55°F. and infection most rapid at 75°F. These conditions are ideally realized when cool, dewy nights are followed by moderate, cloudy days. High summer temperatures and dry weather check the disease. These factors account both for the seasonal prevalence and geographic distribution of the disease, and its late development (July and August) in northern areas. The relation between vigor or succulence of the host and its susceptibility to late blight is not clear. Heavy soils predispose potatoes to late blight, probably through their water-holding capacity which favors both infection and secondary decay of the tubers.

Control: 1. TUBER SELECTION. Planting tubers should be free from late blight lesions, obtained by use of certified or selected seed. Most states include late blight infection as a cause for disqualification of potatoes for seed purposes, and in some of the southern states, where the only source of primary infection is believed to be infected, northern-grown seed tubers, strict seed tuber inspection laws are regarded as the principal defense against late blight:

2. SPRAYING OR DUSTING. Applications of Bordeaux mixture, 4-4-50, are standard for late blight control. Where late blight is a problem, spraying should start when the vines are 6 to 8 in. tall and continue at 10-day intervals until harvest. Applications must be thorough, preferably with a sprayer delivering a fine mist under 200 lb. pressure (Fig. 221), and are best given before, rather than after, rains. The spray repels leaf hoppers and by its shading effect reduces sun injury (tipburn). Instead of spraying, copper-lime dusting is often practiced, using 20 lb. of dehydrated copper sulfate to 80 lb. hydrated lime. It is best applied early in the morning when

the vines are wet with dew. Fixed copper dusts or sprays also are good fungicides, and recently outstanding results in late blight control have been obtained with Dithane-ZnSO₄-lime spray and with Phygon.

Late blight is erratic in appearance, and spraying or dusting for blight may be economical only during years of serious outbreaks. These cannot be foreseen by growers, but the organization in 1942 of the "Late Blight Forecasting Service" has provided a means of warning growers of impending epiphytotics, when prompt spraying is essential to avoid serious crop losses. The service is based on numerous, widely-scattered inspection plots in which the approach of an epiphytotic can be charted from day to day.

For growers with small acreages the cost of spray equipment is a large item in potato production. This has been simplified by the organization in some states of spray rings, in which groups of farmers contract for the services of large spray rigs, reducing the cost of the applications and assuring that the applications will be made properly, at the right times, and with effective materials.

3. CULTURAL METHODS. Growers who are anxious to obtain highest prices for an early-harvested crop sometimes prefer to dig potatoes when the vines are still green. This favors late blight infection of the tubers, but the danger may be avoided by killing the vines with a herbicide, such as Sinox, Dowspray 66 Improved, Ammate, or ammonium thiocyanate, which also makes digging easier, and avoids the development of secondary, off-type, and oversized tubers.

Since diseased sprouts from potato dump piles are dangerous sources of primary infections, destruction of these piles is a very practical way of reducing the disease. In Maine such destruction is required by law. If dump piles are not destroyed they may be made less hazardous by killing the sprouts, as they form in the spring, using one of the herbicides mentioned above.

4. STORAGE. To reduce secondary decay, storage is most satisfactory at 40°F. Tuber treatment of potatoes from infected crops with mercury before storage is recommended in Ireland, but this applies only to tubers saved for seed; it would render them unfit for food.

5. RESISTANT VARIETIES. The rapid progress in development of blight-resistant potatoes, discussed on p. 213, gives increasing opportunity to combat the disease by varietal resistance. It is probably only a matter of a few years before blight-resistant varieties well adapted for each potato region will be generally available.

6. CONTROL OF THE DISEASE IN TOMATOES. In tomatoes the disease is controlled by spraying or dusting just as in potatoes and during

epiphytotics by storing the crop for a few days in dry open sheds and then culling before marketing. Where infected tomatoes are a hazard to either tomato or potato crops, the danger can be avoided by using home-grown or certified tomato plants, grown with regular and thorough seedbed spraying.

DOWNY MILDEW OF CUCURBITS (*Pseudoperonospora cubensis*)

Under some conditions this is an important disease of cucumbers and cantaloupes, and in greenhouse culture (Fig. 113). The foliage undergoes



FIG. 113. Downy mildew of cucumber. (Courtesy, C. J. Nusbaum, S. C. Agr. Exp. Sta.)

rapid necrosis, the lesions bearing sporangia that are wind-borne and may germinate directly or by the production of zoöspores. The disease is favored by cool temperatures (68°F.) and high humidity. It is controlled by weak Bordeaux spraying or, better, copper-lime dusting, and to some extent by resistant varieties such as crosses of a Chinese cucumber with Early Black Diamond, certain Puerto Rican selections and the Texas resistant cantaloupe No. 1.

DOWNY MILDEW (BLUE MOLD) OF SPINACH (*Peronospora effusa*)

This disease occurs in all parts of the United States, and in the mammoth spinach industry of Texas it is a vital production factor. The disease causes yellowing, stunting, distortion, and killing of the leaves in the field,

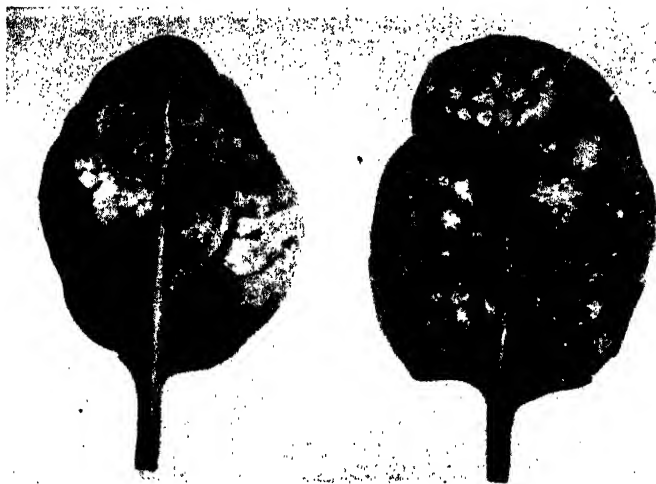


FIG. 114. Spinach leaves severely infected with downy mildew or blue mold, viewed from the under surface. The tip of the leaf on the right shows necrotic spots caused in mildew-infected leaves by a secondary weak parasite, *Heterosporium variable*. (Courtesy, M. A. Richards, Cornell Agr. Exp. Sta., Bull. 718.)

often advancing in a very destructive manner during storage and transit to market (Fig. 114). The fungus hibernates mainly on overwintered spinach plants, thence is spread to the spring crop by wind and rain under cool conditions (45° to 65°F.). Outbreaks of the disease appear to relate directly to practices on individual farms concerned, since heavily infested fields may be found near noninfested ones, which does not indicate any extensive wind dissemination of the disease from one farm to another. In the South, where the disease often is very severe, it is not known with certainty how the disease oversummers. Oöspores of the fungus can be found sometimes in seed from infested fields, and these may be the source of primary infections in the fall or spring. While spinach is the only host, no resistant varieties are known.

Control is aided by separating overwintered crop or volunteer spinach from spring plantings. Spraying is ineffective or impractical, partly because it is disfiguring to the foliage, but mainly because of the difficulty of applying a spray to the under surfaces of the leaves where it is needed. Control by addition of fungicides to irrigation water is promising but still experimental. Such methods introduce the danger of poison residues on the crop, which would eliminate the heavy metals as fungicides for a leafy crop like spinach. Until more is known of the seed transmission of this disease, it is advisable to plant only the highest quality seed obtainable. Reduced planting rates, and drilling the seed rather than broadcasting

it, tend to prevent dense masses of foliage which retain humidity and favor blue mold infection.

DOWNY MILDEW (BLUE MOLD) OF TOBACCO
(*Peronospora tabacina*)

The leading seedbed disease of tobacco is downy mildew. It appears as a bluish-gray mold that overruns the seedbed and in a very short time may ruin thousands of seedlings with a rapid necrosis (Figs. 115, 116). No



FIG. 115. Downy mildew or blue mold of tobacco.
(Photograph, Va. Agr. Exp. Sta.)

zoöspores are produced but instead the sporangia regularly germinate by infection threads. These can be wind-borne for considerable distances. Oöspores are produced in the dead tissues and these serve to overwinter the fungus. The disease is favored by cool temperatures and abundant moisture. This disease can be prevented by spraying or dusting plants in the seedbed. Popular fungicides are yellow or red copper oxide (Cupro-cide) plus cottonseed oil and an emulsifier, bismuth subsalicylate, Dithane, and Fermate. The last is effective also against flea beetles in the seedbed. Both prevention and cure can be effected by the unusual but highly effective practice of fumigating the seedbeds with benzol vapor or with para-

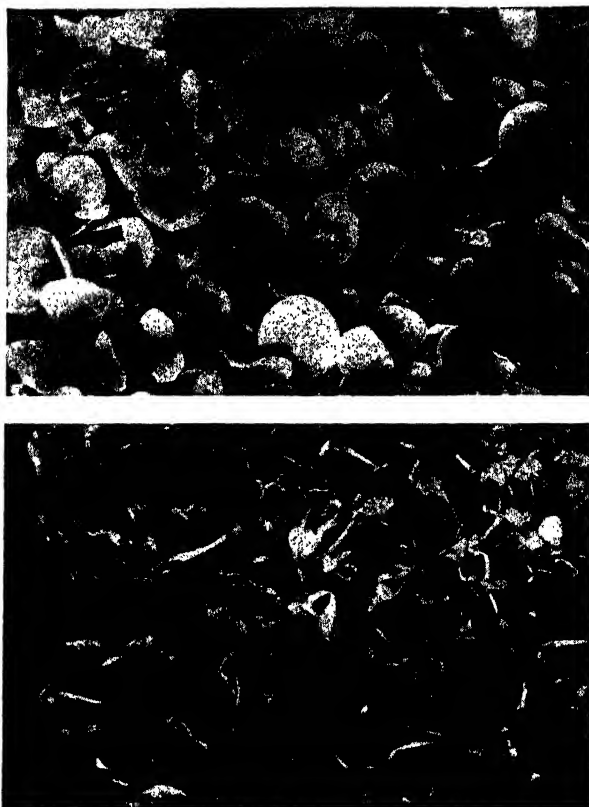


FIG. 116. Downy mildew or blue mold of tobacco. (*Top*) Healthy seedlings. (*Bottom*) A bed severely attacked. (Courtesy, P. J. Anderson, Conn. Agr. Exp. Sta.)

dichlorobenzene (Fig. 224). The benzol is allowed to evaporate overnight from a wick-type evaporator or a flat pan while the paradichlorobenzene is sprinkled on a cloth over the seedlings. In either case the bed is covered with cloth or canvas during the night.

DOWNY MILDEW OF GRAPES (*Plasmopara viticola*)

This has been the most devastating disease in the commercial wine industry of Europe to which it was brought from America on *Phylloxera*-resistant understocks. In America it is most destructive to European varieties. The disease appears on grape leaves as yellowish, then necrotic spots bearing on the under surface a white mold of sporangiophores and sporangia (Fig. 117). Young fruits are very susceptible and drop when infected. Oöspores develop within the leaves, and these, together with mycelium in the buds and crowns, serve to overwinter the fungus. The sporangia germinate by means of zoöspores, most abundant infection

occurring at temperatures between 77° and 83°F. and under humid conditions. Control depends on destroying the fallen leaves by burning and spraying as for black rot control (see p. 109).

DOWNY MILDEWS OF GRAINS AND GRASSES

(*Sclerospora* species)

In moist regions, especially in the tropics, downy mildews of the genus *Sclerospora* are common and often serious on corn, sorghums, millet, sugar

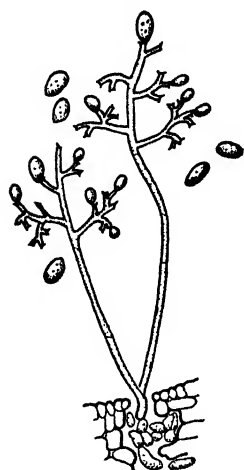


FIG. 117. Sporangio-phores and sporangia of the downy mildew of grape. (After Smith.)

cane, and other *Gramineae*. The diseases are primarily necrotic, with more or less extensive destruction of the foliage. In millets an abnormal stimulation of dormant buds and leafy proliferations of the floral organs follow infections. Sporangia are produced on the necrotic areas although often sparsely, sometimes only at night. The tissues become filled with thick-walled oöspores (*Sclerospora* = "hard spore") that are very resistant to unfavorable conditions and often germinate with difficulty. In the Southwest, during moist seasons, the rare *Sclerospora farlowii* may become abundant on Bermuda grass, causing short black dead areas that prune off the tips of the leaves and do a fairly efficient job of mowing Bermuda grass lawns without any serious damage to the grass (Fig. 118). The control of the downy mildews of cultivated *Gramineae* con-

sists mainly in breaking the sequence of host culture by rotations or fallowing. Sometimes seed treatments are beneficial, and in many cases downy mildew-resistant cereals have been developed.

OTHER DOWNY MILDEWS

Other downy mildews of occasional importance occur on cabbage and other crucifers, alfalfa, clover, carrots and other umbellifers, lettuce, beet, lima bean, pea, onion, violet, pansy, rose, coffee, and cocoa. The downy mildew of hops is the leading disease of this crop in many areas. Stems, blossoms, and cones are attacked and killed, greatly reducing the quality and quantity of the crop. The disease is controlled by regular spraying with Bordeaux mixture, 6-4-100 or Cuprocide-Y, 1½-100, and by treating the hops stakes and twine—sources of infections for nodes and buds—with a fungicide such as Puratized or Phygon. Cabbage downy mildew, which develops like tobacco blue mold in seedbeds, is controlled

by benzol fumigation or fungicidal spraying. The downy mildew of soybeans is held in check by dusting the plants with sulfur.

White Rusts

While closely related to the downy mildews, the white rusts differ from them in that the sporangia are produced in chains within compact sori resembling those of true rusts. The only genus is *Albugo* with about 15 species, chiefly noted as pests of cruciferous plants, spinach, sweet potato, salsify, and pigweed.

WHITE RUST OF CRUCIFERS (*Albugo candida*)

White rust of crucifers is common in plants of the family *Cruciferae*, including cabbage, cauliflower, cress, mustard, horse radish, radish, rutabaga, turnip, salsify, wallflower, stocks, and weeds such as shepherd's purse and pepper grass. Ordinarily it is not very destructive but occasionally it may be serious on mustard greens, turnips, horse radish and radishes. Affected plants show yellow spots on the leaves; eventually, on the under side, white powdery pustules of sporangia burst through the epidermis (Fig. 119). The sporangia are in chains of 5 to 10 spores (Fig. 120). These are wind-carried, and germinate on moist leaves, liberating 6 to 18 zoöspores which, after a swimming period, come to rest and protrude an infection thread which passes down through a stoma and establishes infection. Affected stems frequently are distorted, curled, or swollen, and floral organs may be hypertrophied. As the affected tissues die, thick-walled oöspores often are formed within them which, after a resting period, may germinate, liberating 50 to 100 zoöspores. Overwintering is accomplished by oöspores in the refuse of infested crops and as mycelium in the crowns of perennial crucifers. Several host-specialized races of the fungus have been detected. Disease development occurs



FIG. 118. Downy mildew of Bermuda grass. Leaves much enlarged showing one breaking over, characteristic of the disease, and in the larger leaf, the dark oöspores occupying the diseased tissues.

chiefly at low temperatures that favor dew formation and germination of sporangia (50°F.). Standing water is necessary for spore germination, but once infection is accomplished it progresses under dry conditions. Control measures rarely are called for but, where indicated, should consist of crop rotation, destruction of cruciferous weeds near cultivated crucifers, and destruction of affected crops. Spraying is required only in exceptional



FIG. 119. White rust of mustard. (Courtesy, G. F. Weber, Fla. Agr. Exp. Sta.)

cases. It is not very effective with horse radish in which the perennial crowns become infected.

WHITE RUST OF SPINACH (*Albugo occidentalis*)

This disease was first recorded on spinach from Texas arriving in the New York market in 1937. The following season it was epiphytotic in Texas, proving to be the most destructive spinach disease of the year, being present in every field examined, and reducing the huge Texas crop by one fourth. It occurs also in Oklahoma and Arkansas. The white, powdery spore masses resemble those of *A. candida*; later, the leaves

are killed. The parasite was known previously only on the goosefoot or pigweed, *Chenopodium capitatum*, which occurs in North Texas, and from which evidently it had spread to spinach. The fungus appears to hibernate in overwintering spinach and to spread thence to the spring-planted crop. When 120 spinach varieties were tested for resistance the damage varied from 10 per cent to 90 per cent in different varieties. Of the important varieties, Bloomsdale Long Standing was much more severely affected than Viroflay, many fields of which yielded normal crops. Other varieties with less than 20 per cent damage were Broad Flanders, King of Denmark, Victoria, Zwann's Dark Green Bloomsdale, Zwann's Darkie, Prickly

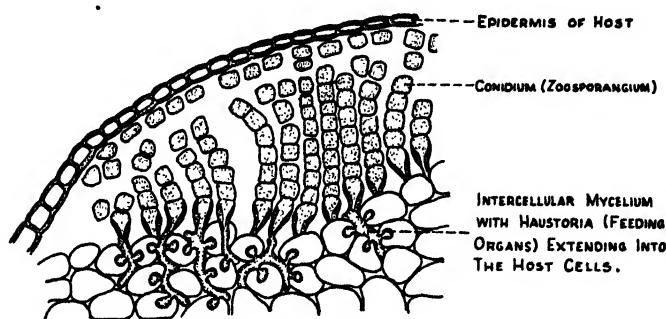


FIG. 120. Reproduction in white rust of crucifers. Section through a fruiting lesion showing mycelium and spores.

Winter, Harlem Market, and Dark Green Giant Prickly. Control, so far as present knowledge goes, depends on separating overwintering spinach from the spring crop, and the use of the more resistant varieties.

Bread Mold

SWEET POTATO SOFT ROT AND STRAWBERRY LEAK (*Rhizopus nigricans*)

Rhizopus nigricans is the black bread mold of which the coarse, cobwebby mycelium and little black balls of spores are familiar to everyone. The fungus primarily is a saprophyte, but under some conditions it can actively invade plant tissues. As a pathogen it is best known for its ability to cause rot of storage organs, especially fruits, although it is capable of producing rapid necrosis of leaves in saturated air. The decays of sweet potato (soft rot or ring rot) and strawberry (leak) are the best known of the *Rhizopus* diseases.

History and Distribution. *Rhizopus nigricans* is of worldwide distribution, but has been recognized as a pathogen of sweet potatoes and

strawberries only since 1914. Since that time it has attained major importance as cause of storage, transit, and market losses with these and other crops, especially when grown in the South and shipped to the northern states.

Importance. Cars of sweet potatoes reaching the Chicago and New York markets show from 1 to 70 per cent infection with *Rhizopus* rot, the average infection being about 10 per cent and the loss in individual cars sometimes exceeding 25 per cent. Leak is practically always present in overripe, bruised, or improperly refrigerated strawberries with many market lots showing 10 to 20 per cent infection.

Rhizopus rot ranks with brown rot as one of the two most serious market diseases of peaches. Occasionally cars may show 25 per cent loss from this cause. It is destructive, with losses up to 20 per cent, in overripe tomatoes, especially those shipped from Mexico, Cuba, and other distant points. In peppers and pimentos *Rhizopus nigricans* is the most important cause of market decay, with market losses recorded from 3 to 30 per cent. *Rhizopus* rots are common in grapes, beans, and cucumbers, and occasionally serious in avocado, pineapple, prunes, and prickly pear. These losses are taken from data in the *Plant Disease Reporter*, referring only to the losses of the distributor and ultimate consumer. In addition, there are the considerable losses experienced by the grower between harvest and marketing, and by the shipper. Nor should we forget the important destruction of plant products, especially prepared foods, caused by this ever-present fungus.

Host Plants. The crops mentioned above are only a few of the more important plants affected by *R. nigricans*. Almost any kind of stored fruits, vegetables, and field crops can be attacked under storage conditions favorable to the fungus. It is associated also with root decay or damping-off of many kinds of plants, and is one of the causes of cotton boll rot.

Symptoms and Signs. In all plants affected, the principal symptom is a rapid, soft decay. In sweet potatoes the roots are not markedly discolored. While the skin remains intact, the internal tissues become mushy and stringy, and watery exudate often wets adjacent roots. Frequently, the end of the potato may be attacked (soft rot) or the rot may form a depressed belt around the root, referred to as ring rot—a term also applied to the rot of sweet potato caused by another phycomycete, *Pythium ultimum*. Ultimately the potato shrivels and dries to form a mummy. Under very moist conditions, the coarse white mycelium protrudes through ruptures in the skin, bearing erect sporangiophores surmounted by sporangia containing black sporangiospores (Figs. 111, 121).

On strawberries and grapes, *Rhizopus* rot is often called "leak." It is a

rapid decay, the affected fruit being overrun with the familiar *Rhizopus* mycelium and dark sporangia.

The signs of *Rhizopus* decay include the odor of fermentation, described in sweet potato as being at first yeastlike, later-resembling the odor of wild rose or geranium. In apples and peaches, and in the later stages of decay in sweet potatoes, the tissues are darkly discolored, but this does not occur in strawberries and grapes.

Etiology. The rots considered here are ascribed generally to *Rhizopus nigricans*, although sometimes other varieties or species of *Rhizopus* are involved. In the case of sweet potato, nine *Rhizopus* species cause similar soft rots, although the majority are due to *R. nigricans* operating at cool temperatures and *R. tritici* which is favored by warmth.

Rhizopus is a genus of higher phycomycetes exhibiting nonsexual reproduction by means of air-borne sporangiospores and sexual reproduction through the union of similar gametes. The vegetative mycelium is coarse, with relatively few cross walls, and is filled with granular protoplasm and vacuoles that can easily be seen streaming along the hyphal stretches. The hyphae assume three habits: there are rootlike feeding hyphae buried in the host tissue, erect hyphae (sporangiophores) tipped with the spore-filled sporangia, and horizontal stoloniferous hyphae that reach out laterally, "strike root," and spread the mycelium in vegetative fashion, much like the runners of strawberries. The sporangium is a round spore case with fragile wall, containing great numbers of tiny, dark sporangiospores surrounding the swollen apex of the sporangiophore. At maturity the sporangial wall weathers away, releasing the spores (Fig. 111).

Occasionally sexual reproduction occurs. The fungus is *heterothallic*, i.e., two sexes of mycelium are found, designated as + and -. When hyphae of these two sexes come in contact, each produces a short side branch. The + and - side branches unite, each cuts off a reproductive



FIG. 121. Soft rot or ring rot of sweet potato. If the skin is ruptured, the mycelium develops on the surface producing numerous sporangia. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

cell (gamete) and the two gametes fuse to form a thick-walled sexual *zygospore*, as illustrated in Fig. 111. Usually this stage is not seen in nature but it may be demonstrated easily by "planting" a culture dish of nutrient agar with the + and - strains of *R. nigricans*.

The sexual stage appears to play little part in the natural distribution of the fungus. In contrast, the sporangiospores are produced in enormous numbers, so much so that the air everywhere constantly contains them, and susceptible substrates develop growths of *Rhizopus* with great regularity when exposed to the air. It is no wonder, then, that bruised fruits and vegetables held in environments favorable to the fungus, regularly develop *Rhizopus* infection from the omnipresent air-borne spores.

Epiphytology. Infections ordinarily take place through fresh wounds. The various *Rhizopus* species that produce storage decay flourish at different temperatures. There is a high temperature group with optimal development at 90° to 95°F., and a low temperature group including *R. nigricans* that develop best from 68° to 75°F. The extremes of temperature between which any of these species can operate are 38°F. and 108°F.

Saturated air or condensed moisture are not necessary for *Rhizopus* development; in fact, at 74°F. sweet potatoes show a higher percentage of infection at 75 to 84 per cent relative humidity than in nearly saturated air.

Control. The details of *Rhizopus* control differ with the crop but similar principles apply to all crops.

1. **STORAGE CONDITIONS.** The leading single factor in preventing *Rhizopus* decay of sweet potatoes is to maintain in storage a high humidity and a temperature that favors corking over of wounds. The recommended temperature is 55° to 60°F. after curing from 10 days to two weeks at 80° to 85°F. Strawberries are shipped best at or below 50°F.; the *Rhizopus* hazard begins to rise two to three degrees above this. Strawberries should be picked in the cool part of the day and refrigerated and shipped without delay. Efforts should be made to avoid wounding potatoes; rat control will help in this.

2. **SANITATION.** It is futile to hope to create a harvesting and storage environment free from *Rhizopus* spores since the fungus is of universal occurrence. This is no excuse, however, for increasing the concentration of spores by allowing moldy heaps of cull fruits and vegetables to lie about. Proper sanitary measures to lessen the danger of this and other storage decays include disposing of culls, occasional disinfection of storage houses and containers, and general cleanliness about the packing shed and storage house. Disinfection may be effected by whitewashing walls, bins, and floor, by spraying the house with copper sulfate, 1 lb. in

25 gal. of water, or by burning sulfur, 1 lb. per 1000 cubic feet of space. The gas generated by pouring 3 pints of formaldehyde on 23 oz. of potassium permanganate for each 1000 cu. ft. is a good disinfestant but care must be used to avoid inhaling the irritating gas. Dipping the potatoes in borax solutions before storage controls soft rot but may burn the potatoes and is discouraged by the Pure Food and Drug Administration.

3. **VARIETAL RESISTANCE.** Sweet potato varieties differ in their susceptibility to *Rhizopus* decay. The most susceptible varieties are Gold Skin, Yellow Jersey, Belmont, Red Brazil, Haiti, Yellow Yam, and Dooley. The more resistant varieties are Nancy Hall and Southern Queen. Varieties intermediate in their resistance include Porto Rico, Big Stem Jersey, Triumph, Pierson, Florida, and Dahomey.

Chytrids

BROWN SPOT OF CORN (*Physoderma zeae-maydis*)

History and Distribution. Brown spot, also known as measles, pox, and dropsy of corn, is primarily a disease of the moister southern states, from the eastern halves of Kansas, Oklahoma and Texas eastward, and extending north with less destructiveness to Minnesota and New Jersey. It occurs also in India, China, and Japan. The disease was first noted in the United States in 1912 but had doubtless been present prior to that time. It was extensively studied first by Tisdale in 1919, and later important contributions to our knowledge of the disease were those of Eddins and Voorhees in 1933-1935 regarding the relation of environment to brown spot and its control by varietal resistance.

Importance. Brown spot is not regarded as a major corn disease in the corn belt, where it rarely causes important losses. In the warm, moist southern and subtropical areas, however, it frequently becomes destructive, causing losses up to 10 per cent. Even as the drier prairies are approached, we find occasional instances of sufficient damage by brown spot to arouse the concern of growers. The loss is due to devitalizing of the plants and to lodging caused by the disease.

Host Plants. Corn and the closely related teosinte are the only hosts of *Physoderma zeae-maydis*. While all corn varieties are susceptible to some extent, they differ in their degrees of susceptibility, and workers in Florida have had success in reducing the severity of the disease by use of resistant inbred lines of corn.

Symptoms. Brown spot is most apparent in the leaf sheaths just above the nodes, although it also occurs on leaf blades and stalks (Fig. 122). The spots are small, 1 to 5 mm. in diameter, rounded, and numerous,

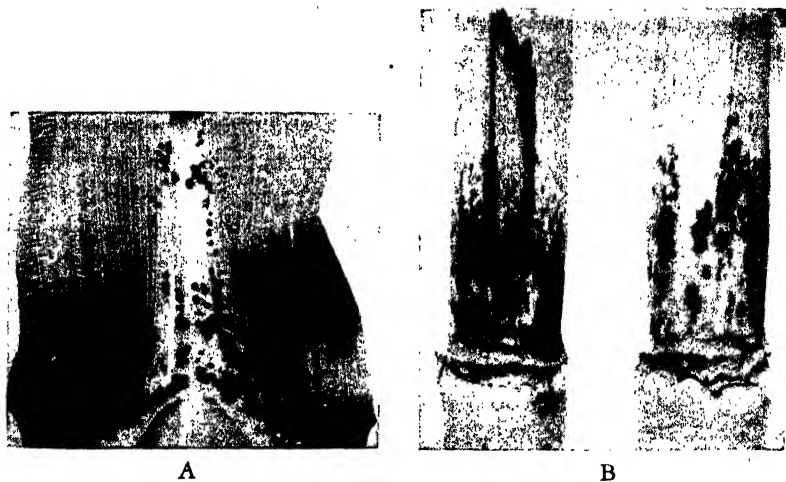


FIG. 122. Brown spot of corn. (A) Upper surface of affected leaf base. (B) Affected stalks, just above the nodes. Both views illustrate the tendency of brown spot to develop in the moist funnels between leaf base and stalk. (Photograph, La. Agr. Exp. Sta.)

at first water-soaked, then bright yellow, and finally chocolate brown. Many may fuse to form large irregular dead areas at the base of the leaf sheath. The spots are most evident on the inner face of the sheath. This position of the injury, when superficially examined, might be confused with the work of chinch bugs or aphids that often congregate in the space between leaf sheath and stem. As the lesions mature, the epidermis ruptures, liberating powdery masses of sporelike sporangia. At this stage the disease resembles a rust. Ultimately the sheath may dry and shred and the stalk may break over at an infected node. A certain amount of abnormal reddening of leaves, sheaths, and stalks commonly accompanies the appearance of lesions.

Etiology. *Physoderma zeae-maydis* is one of the chytrids, the most primitive group of phycomycetes. The brown sporelike bodies, which are formed inside the infected corn cells, are in reality sporangia. Each sporangium is equipped with a circular lid, like a trap-door, and this structure distinguishes these sporangia from spores of other fungi (Fig. 123).

By means of these sporangia, which are highly resistant to unfavorable environments, the fungus overwinters in refuse from a preceding corn crop. In the spring moisture the overwintered sporangia germinate; the lid is detached, and the contents of the sporangium, a protoplasmic mass, oozes out, and at once separates into individual one-celled zoöspores. The zoöspores swim about actively, being each equipped with a long

whiplike cilium. Ultimately the zoöspore comes to rest, absorbs its cilium, and becomes amebalike. It then extends a fiberlike hypha which is able to penetrate the corn epidermis through a stoma or cuticle and invade the epidermal cells. Instead of germinating by means of a fiberlike hypha, the zoöspores occasionally come to rest and produce a slipper-shaped sporangium in place of a hypha. This thin-walled sporangium liberates about 300 smaller zoöspores of uncertain function.

Within the infected host cell there is formed an enlargement of the infecting fiber, and from this, new fibers pass out to nearby cells and repeat the process. Within each infected cell the swollen hyphae become transformed into the brown, thick-walled resting sporangia, several to a

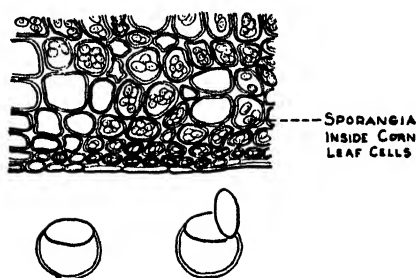


FIG. 123. Brown spot of corn. (Top) Appearance of infected corn tissues. (Bottom) Sporangia showing the characteristic lid.

cell, and these are liberated from the dead host cells as the tissues decompose or the epidermis weathers away.

Epiphytology. The resting sporangia are highly resistant to physical and chemical agencies. They can survive one or possibly two winters in the field, and can withstand freezing temperatures ranging from 32° to 18°F. They are resistant also to digestive fluids and can pass through the alimentary tract of farm animals without injury. Germination of the sporangia requires saturated air, as is often found between leaf sheath and stem, and proceeds best at 82°F. They will not germinate under 74°F. Twelve to 48 hours are required for germination. The combined temperature and moisture requirements are responsible for the southeastern and subtropical range of *P. zeae-maydis*. The sporangia are spread primarily by wind, aided by rain and surface water and the movement of soil.

Control. No extensive studies on the control of brown spot have been made. Judging by the nature of the disease, it should be reduced in severity by (a) rotation with one or preferably two years intervening between corn crops, (b) early, deep, and thorough plowing to dispose of

infested corn debris, and (c) avoidance of using manure for corn land from animals fed on infested corn stalks. These are the same measures as used for controlling the common corn smut, which doubles their value. At the Florida Agricultural Experiment Station certain inbred lines of corn have been selected as resistant to brown spot, but no resistant varieties or hybrids are yet available commercially.

CLUBROOT OF CRUCIFERS (*Plasmodiophora brassicae*)

History and Distribution. Clubroot, also known as finger-and-toe disease, hernia, and clubbing, is a classic plant disease in many respects. Although first recorded in England in 1736, it probably existed in Europe much earlier than that. It appears to be the same trouble that was known as "cabbage syphilis" at the beginning of the fifteenth century in Spain. During the 1700's it caused much injury to turnips in Scotland. It was a problem in the United States by the middle of the nineteenth century, but prior to Voronin's masterful study in 1873-1876, the cause of clubroot was unknown, and it was variously attributed to insects, excessive manure, reversion to wild types, or environmental disturbances. By 1872 clubroot was so destructive in Russia that a prize was offered by the Russian Gardening Society for its solution. Independently of this inducement, the brilliant Russian scientist, Voronin, in 1873 began an intensive study of clubroot, and in 1878 he published such a complete account of the life history of *Plasmodiophora brassicae* that comparatively little has been added in spite of intensive subsequent researches. Voronin's paper, which every student of this disease should consult, is available as *Phytopathological Classic 4* (1934). More recent researches, particularly in the United States, Russia, England, and Germany, have been especially concerned with doubtful points in the life history of *P. brassicae* and control of the disease by soil treatments and the use of varietal resistance.

The present distribution of clubroot includes nearly all parts of Europe where cruciferous crops are important—the United States, Canada, Alaska, Australia, New Zealand, South Africa, and India. In the United States it occurs in 36 states and is important in 21 of them.

Importance. The losses from clubroot are very considerable but difficult to estimate. Infestation in the seedbed produces seedlings that are worthless as transplants. In the field the root injury may lead to partial or complete crop loss, cabbages failing to head, and root-crop crucifers exhibiting stunting, yellowing, wilt, absence of fleshy root, malformation, and premature death. Added to the direct damage due to water shortage is the decay that regularly follows clubroot infection. There may be a loss also from the fact that once land is infested, susceptible crucifers cannot

be safely grown on that land for many years. This is particularly important because of the peculiar soil requirements of some of the crucifers, notably cabbage.

Host Plants. Clubroot occurs only on plants of the *Cruciferae*, wild and cultivated. Within this large family there are many highly susceptible species and a few that are immune. In general, nearly all of the cultivated cruciferous crops are highly susceptible: cabbage, radishes, cauliflower, brussels sprouts, rape, mustards, alyssum, and pepper grass. Among the cultivated varieties of turnips and rutabagas are numerous resistant varieties, e.g., yellow, firm-fleshed, rutabaga types; Early White Milan, Early Snowball, Yellow Stone, Yellow Egg, Sweet German, White Swede, Sweet Russian, Purple Aberdeen, Yellow Rutabaga.

Resistant turnip and rutabaga varieties are susceptible in some areas, evidently due to differences in physiologic races of the clubroot fungus. No varieties of cabbage, cauliflower, kohlrabi, or brussels sprouts have any practically important resistance.

Symptoms and Signs. Clubroot may be apparent at any stage of the host's development, from seedling stage until the first frost. Above ground, infected plants exhibit symptoms of chronic water deficiency, wilting during hot, sunny days with recovery at night, yellowing of the outer leaves, and failure of cabbages to head, these symptoms often being followed by premature death. When such plants are pulled up, the root system is found to be a grossly distorted mass of large and small irregular swellings (Fig. 124). There may be a single massive gall, or more often several sweet-potato-shaped galls, the reason for the name "finger-and-toe." Either the tap root or the lateral roots or both may be affected. The swellings often are associated with more or less extensive decay, or the infection may result in scabbing, fissuring, or decomposition of the roots. The only other trouble likely to be confused with clubroot is the nematode disease, root knot. (See Fig. 176.) While the two troubles are similar superficially, root knot can be distinguished by the presence of the pearly female nematodes buried in the tissues. Moreover, clubroot is limited to cool growing conditions while root knot is a high-temperature disease. Rarely, similar swellings are produced by root-feeding insects or genetic abnormalities.

The swollen roots are not discolored nor hollow, but are white and firm until secondary decay occurs. Microscopic sections through an infected root show that the water-conducting xylem elements are very poorly developed, and here and there are seen groups of very large, dark-staining cells filled with the protoplasmic mass or tiny spores of the pathogen.



FIG. 124. Club root of cabbage. Compare with Figs. 150 and 176. (Courtesy, C. T. Gregory, Ind. Agr. Extension Serv.)

Etiology. *Plasmodiophora brassicae* formerly was held to be a myxomycete or slime mold but now it is considered to be a fungus of the primitive group of chytrids, kin to the corn brown spot organism.

It persists between crops, often for many years, as resting spores in the soil. Alternate periods of freezing and thawing do not kill them but even aid their later germination. In the spring, with the occurrence of favorable conditions of temperature, moisture, and soil, the resting spores germinate, each producing a motile swarm spore. At first these are equipped with a flagellum but soon this is absorbed, and the swarm spore becomes amebalike, moving about by means of protoplasmic streaming. On reaching a root hair or other suitable infection court the ameboid swarm spore penetrates into the host tissues. Root hairs generally are the portals of entry, although wounds or even uninjured cuticle may serve for points of infection. The ameba passes down to the base of the root hair and by penetration to adjacent cells and, by division of the amebas and division of infected cells, a large number of infected cells results. According to one

account, the amebas in the root hairs become sporangia, liberating four motile zoöspores which go through the sexual stage of fusing in pairs before beginning their general invasion of the root. Meanwhile the number of nuclei in each swarm spore is greatly increased. After a period of occupation of a host cell by the fungus, its protoplasm collects about each nucleus, which then rounds off and becomes a minute resting spore, thousands in a single cell. These have no special means of distribution, but finally are returned to the soil with the decomposition of the root.

During invasion of the root the host cells are stimulated to excessive development, food materials are drained away from the parts above ground and the swollen clubs result.

Epiphytology. Temperature, moisture, soil reaction, and aeration are important in the development of clubroot. Disease development occurs between 54°F. and 80°F. with maximum at 77°F. Spores germinate between 43°F. and 80°F. with a maximum also at 77°F. Clubroot develops best at temperatures somewhat higher than optimum for host root development. Soil moisture is even more important than temperature, with infection taking place between 50 and 100 per cent moisture-holding capacity of the soil, particularly at the higher points. Thus, rainy seasons and poorly drained soils predispose plants to clubroot. Soil reaction is a limiting factor, the disease occurring only in neutral to acid soils with a pH ranging between 5.0 and 7.0. There is less infection in humus-rich, well-aerated soils than in heavier soils.

Spread of the disease is brought about by any means of moving infested soil, manure, or plant refuse, or by drainage water from infested fields. Movement of infected seedlings serves principally for long-distance spread. Wind dissemination is unimportant except in very light soils.

Control: 1. **SANITATION.** As clubroot usually is introduced into new areas with infected seedlings, careful inspection and supervision of seedlings and their source is indicated. The refuse from infected crops is another important danger and should be disposed of in such a way as to avoid future soil contamination. The resting spores pass through animal bodies uninjured, resulting in the hazard of using infested manure. Where infested and uninfested fields are close together the grower must be alert to the possibility of transferring the pathogen from one field to another on machines, tools, and feet.

2. **ROTATION.** Crop rotations would have to extend over a very long time to be of value in clubroot control, and badly infested land is better abandoned for production of susceptible crops.

3. **SOIL DISINFESTATION.** Where danger of soil infestation exists, seedbeds can be rendered safe by chemical soil disinfestation, as with corrosive



FIG. 125. The slime mold, *Mucilago spongiosa*, while not a true parasite, becomes troublesome by climbing over and smothering young shoots of various plants. Here it is seen on strawberry, which frequently is overrun by the slime mold in this fashion.

sublimite, 1 oz. in 10 gal. of water, or with Semesan. Heating soil to boiling temperature is effective also. In the field, liming to bring the soil at least to pH 7.2 has long been recognized as the chief preventative of clubroot if this is compatible with culture of other crops in the rotation. Hydrated lime is most effective but other forms can be used, the dosage varying with the pH and character of the soil. At the same time the use of barnyard manure or superphosphate increases the disease. The striking effects of lime in clubroot control were brought out in Vermont where the yield of cabbage was increased from 672 lbs. to 23,182 lbs. per acre following liming. Failure of liming to give complete control may be due to local, more highly acid spots in the field, or to an acid film of moisture about the roots caused by solution of CO_2 , given off by the roots, in soil moisture.

Mercury soil treatment has given good results as an accessory to other control measures. After setting, each plant is watered with $\frac{1}{3}$ pint of mercuric chloride, 1:1500, or calomel or yellow mercuric oxide is mixed with fertilizer and applied to the field at the rate of 5 to 12 lbs. per acre.

4. DRAINAGE. Drainage and lightening of the soil are accessory means of lessening club root damage.

5. VARIETAL RESISTANCE. It has been pointed out that certain varieties of turnips and rutabagas are resistant to clubroot. These may be given preference. Physiologic specialization in *P. brassicae* indicates the possibility that under some conditions they might prove susceptible.

Slime Molds

The clubroot organism formerly was classified as a slime mold. Today it is considered a phycomycete and this leaves the slime molds with very few members of pathologic importance.

The slime molds or myxomycetes have an amebalike feeding stage, and reproduce by heaping up slimy masses of protoplasm which become converted into spores. One of the group, *Mucilago spongiosa*, has the habit of climbing over the foliage of strawberries, grasses, and nursery cuttings. It is recognized as large, oval, powdery, white, later steel-gray spore masses (Fig. 125). Under moist conditions it can be quite a nuisance and cause injury by smothering the plants. No information is available on its control, but in all probability a dusting or spraying with sulfur or Bordeaux mixture would be effective as a repellent.

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Chapter 9

Damping-off and Related Troubles

Damping-off (*Pythium debaryanum*, *Rhizoctonia solani*, etc.)

From time to time growers of every type of crop are confronted with the problem of poor stands with need of replanting, often at a less favorable date than the original planting, or with a resultant poor yield from broken stands and weakened plants. Poor stands may be due to the use of seed of low viability or to the direct effects of adverse weather, but in most cases they are the result of invasion and destruction of the seedlings by soil pathogens operating under the influence of excessive soil moisture and temperatures unsuitable for best seedling development.

Economic Importance. Estimates of losses from damping-off are not available but many isolated observations of damping-off in nurseries, seedbeds, greenhouses, and fields, and in many crops, indicate the great destructiveness of damping-off. Losses of 50 to 100 per cent in plantings are common experiences to every grower, and many cases might be cited comparable to Horsfall's account of the complete destruction of 200,000 tomato transplants nearly ready for sale at \$1000.

The losses from damping-off take various forms: destruction of saleable seedlings, cost of replanting, loss from replanting at unfavorable late dates, loss from broken stands, poor yields from surviving but injured plants, and consequent effects of the damping-off organisms such as stalk, ear, boll, and fruit rots, and various leaf and stem diseases.

Host Plants. Almost any type of plant may be affected by damping-off but some crops suffer much more than others. Among the more susceptible crops are spinach, legumes, cruciferous vegetables, tomatoes, salsify, cucurbits, beets, cotton, tobacco, corn, sorghums, and forest tree seedlings both coniferous and broad-leaved. Plants are susceptible to damping-off only in the seedling stage, and the importance of the disease in nursery stock relates to the long seedling period in woody plants. Cuttings suffer particularly from damping-off because of their retarded growth rates and wounded stems.

Symptoms: 1. GERMINATION FAILURE. In the early stages of germination the swelling seed may be invaded by damping-off organisms and

destroyed without sprouting. In some cases seed decay is due to seed-borne damping-off organisms, in others it is the effect of soil-borne pathogens. Under conditions favorable to damping-off even the best of seed may fail in this way.

2. **PRE-EMERGENCE DAMPING-OFF.** Before the young seedling reaches the sunlight, as it pushes upward through the soil, it is highly susceptible to damping-off attack. At this time it may be destroyed by swift decay. The failure to get a good "come-up" does not necessarily indicate poor seed, although the grower often blames stand failures on the seed (Figs. 126, 215).

3. **POST-EMERGENCE DAMPING-OFF.** The most obvious manifestation of damping-off is the toppling over and death of seedlings after they have emerged from the soil. Soft, succulent seedlings usually show a water-soaked zone at the soil level; this soon becomes necrotic and shrunk and the plant falls over, often before it wilts. With woody seedlings the plant remains erect but wilts and dies, following the development of a dark necrotic lesion at the soil line. More or less extensive decay of the root system is seen in either case. If the stem is not completely girdled by the lesion, the plant survives, but the injurious effect of the lesion remains and the plant is often stunted and injured for life. This condition in cotton is termed "sore shin."

Damping-off, like other soil-borne diseases, tends to occur in well-defined, ever-widening spots in the planting, the spot containing only affected plants and bordered abruptly by healthy ones. Many of these spots may give the planting a "moth-eaten" appearance. The spots appear very suddenly and spread so rapidly that no more than two or three days

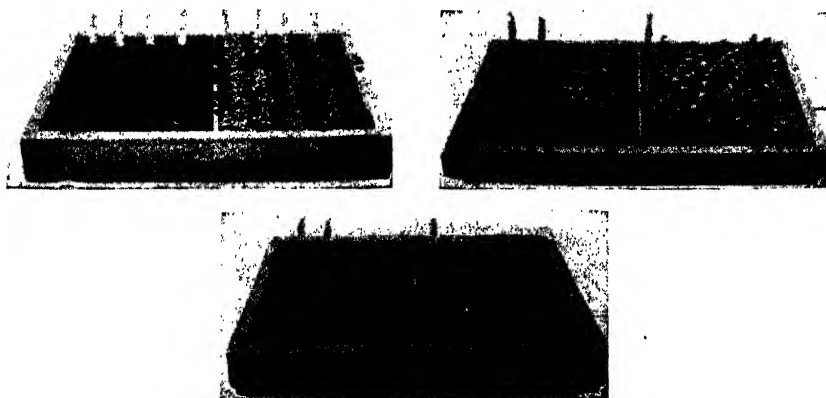


FIG. 126. Damping-off and its control by soil treatment with formaldehyde dust. The soil in the right half of each flat has been treated. The left halves are untreated checks. (Courtesy, P. E. Tilford, Ohio Agr. Exp. Sta.)

may elapse between the first appearance of the disease and the total destruction of extensive plantings.

Etiology. Damping-off may be caused by any one of a number of different fungi, the principal ones being:

1. *Pythium debaryanum*, *P. ultimum*, *P. aphanidermatum*, *P. irregulare*, and certain species of *Phytophthora*, all closely related phycomycetes that are common inhabitants of soil, reproducing by swimming zoöspores and by thick-walled resistant oöspores (Fig. 110).

2. *Rhizoctonia solani*, the same fungus that produces black scurf of potato, described on pp. 88-93, the imperfect, sporeless stage of the crustlike basidiomycete, *Corticium vagum* (Fig. 129).

3. *Fusarium moniliforme* and other *Fusarium* species, imperfect fungi with oval microconidia, banana-shaped macroconidia, and thick-walled chlamydospores (Fig. 129).

4. *Botrytis* species, imperfect fungi with egglike conidia on branched conidiophores resembling bunches of grapes.

5. *Macrophomina phaseoli*, a black, sporeless, imperfect soil fungus persisting by means of tiny, round, black sclerotia.

Of these, *Pythium debaryanum*, which flourishes principally at high soil temperatures, and *Rhizoctonia solani*, which prefers cool soil, usually are considered the most important.

All the various damping-off organisms are facultative saprophytes, and are so successful in their saprophytic existence in the soil that they may be found with great regularity in almost all soils that have not been disinfested. With the exception of some of the species of *Fusarium*, these damping-off fungi ordinarily are not seed-borne, but between crops persist in the soil or the plant refuse that it contains.

Plants usually are susceptible only in the seedling stage but during this period they may readily be invaded and destroyed with a rapid necrosis by mycelium of one or another of the damping-off fungi. The damping-off fungi produce highly active enzymes that break down the various cell constituents, converting them into substances that can be assimilated by the pathogen. Wounds ordinarily are not necessary for fungus penetration.

Overwintering of the damping-off organisms and their persistence in the soil for long periods is accomplished by resting bodies in the soil, oöspores in *Pythium* and *Phytophthora*, sclerotia in *Rhizoctonia* and *Macrophomina*, and chlamydospores or conidia in *Fusarium*, or as active, vegetating mycelium. Mycelium plays the principal role in infection and spread of damping-off. Spores have only a minor part in dissemination of these fungi, and in some cases they are lacking entirely. Spread of the damping-off fungi from one field or planting to another depends largely on the movement of infested soil as on machinery or feet or clinging to transplants.

Damping-off caused by fungi must be distinguished from physiogenic damping-off, a condition that closely resembles fungus attack but is caused by unfavorable environment, particularly burning of young stems from excessive heat at the soil line (heat canker, see p. 386), excess soil solutes, and waterlogging of roots leading to their death from lack of oxygen.

Epiphytology. Most natural soils contain damping-off fungi, but the disease is highly dependent on a favorable environment and appears only when conditions of moisture, temperature, and soil reaction are suitable. The damping-off fungi are active over a wide range of temperatures; in general, the most severe damping-off occurs at temperatures at which the ratio of host plant growth to pathogen growth is low, and less severe when this ratio is high. High soil moisture, from 50 per cent upward, favors the disease. A sudden change from moist to dry conditions sometimes abruptly checks the trouble. Damping-off occurs in soils of various types and reactions as might be expected of a trouble due to any of several organisms, some of which are rather tolerant of environmental extremes. In general, the disease is most severe in heavy, poorly drained soils and in well-fertilized soils. In heavy clay soils that crust over easily, damping-off sometimes is so severe as to lead to complete stand failure. It is suggested that in such soils the seedlings usually are physically too weak to break through the crust, but preëmergence damping-off of the retarded seedlings in the poorly aerated, moist zone under the crust is more likely the principal cause of this type of damping-off. The problem of soil fertility in relation to damping-off must be considered also as a biological problem. The organic material of the soil favors the saprophytic life of the damping-off fungi as well as that of other soil organisms, some of which are known to be antagonistic to *Pythium* and *Rhizoctonia*. Soil sterilization raises a special problem, where the removal of antagonistic soil inhabitants opens the way for unrestricted and deadly development of damping-off fungi when such soils become reinfested. This whole problem of soil competition as it affects the damping-off fungi is almost a virgin field for further productive study and experiment.

Control. Control of damping-off is approached primarily in three ways: the use of treatments to surround the seed with a protectant coating of disinfestant chemical, soil disinfestation, and soil management. There is little information on the resistance of various crop varieties against damping-off. Different seed lots of the same crop variety show different degrees of damping-off, owing mainly to the vitality of each seed lot and its capacity for producing vigorous seedlings.

1. **SEED TREATMENTS.** Most damping-off fungi are soil-borne, not seed-borne. Seed treatments for damping-off control rarely are intended to

kill inoculum on or in the seed, but instead are used with the purpose of surrounding the seed with a chemical that disinfests a small volume of soil about the seed after it is planted, giving the seed a temporary protection against soil-borne fungi. To accomplish this it is necessary to use a chemical that is not washed off after the treatment, as is done with corrosive sublimate. Also, it is desirable that the chemical be slowly volatile or relatively insoluble, so that it will pass out from the seed and effectively destroy or repel soil pathogens in the vicinity of the seed. A number of chemicals meet these requirements, in particular the organic mercury dusts (Ceresan, Semesan, Barbak, DuBay 1452F, etc.), compounds of copper and zinc (Cuprocide, COCS, Vasco-4, Dow 9, etc.), carbonyl compounds (Spergon, Phygon, etc.), chlorinated compounds (Dow 6, Dow 10, etc.), and thiocarbamates (Arasan, Tersan, Fermate, etc.). These perform differently on the various crops. On the basis of local tests the various states issue recommendations of seed disinfestants that are particularly suitable, and for up-to-date, locally applicable information growers should secure these recommendations.

For small seed lots the dusts are shaken up with the seed in a bottle or can. For larger quantities, use commercial treaters or one of those shown in Fig. 19. More and more seed is now being pretreated by the seed processor before sale, an inexpensive and very desirable means of assuring a good supply of properly treated seed. Further details of seed treatments are given in Chapter 20. In legume seed treatments, certain of these chemicals may interfere with bacterial nodulation, but it remains to be seen whether this is a serious handicap or one that cannot be overcome.

Seed treatments for damping-off control are most effective under conditions of moderate disease occurrence (moderate "inoculum potential"). Where the disease is very severe, even seedlings from treated seed will succumb, and where there is little evidence of damping-off there may be no advantage seen in the plants from treated seed.

2. **SOIL DISINFESTATION.** Soil disinfestation for control of damping-off and other diseases is discussed in much more detail in Chapter 20. Soil disinfestation with steam or dry heat should be a routine practice in controlling damping-off in greenhouses and small seedbeds. Where heat is not available, formaldehyde, tear gas, organic compounds such as chloropicrin (Larvacide), or copper and mercury compounds may be used (Figs. 126, 215). Soil disinfestation for damping-off control is practical only on small areas of soil.

3. **POSTEMERGENCE TREATMENTS.** Control of damping-off is mainly a program of prevention. Little can be done to save a crop once the disease has appeared though valuable seedlings in the early stages of damping-off

can be salvaged sometimes by watering the seedbeds with a copper oxide, zinc oxide, or organic mercury solution.

4. **ACIDIFYING THE SOIL.** In forest nurseries the problem of damping-off is a very serious one. Good results in control have been obtained by drenching the nursery bed soil with sulfuric acid, acetic acid, or ferrous or aluminum sulfate, which have acid reactions. Extreme care must be used in these practices because of the danger that seedlings will be unable to grow normally in highly acid soil; rose cuttings, for example, are harmed by this method of damping-off control.

5. **GREENHOUSE AND SEEDBED MANAGEMENT.** We have seen the strict dependence of damping-off on excessive soil moisture. Much can be accomplished in damping-off control by regulating the water supply to avoid excessive soil moisture, and the value of seed or soil treatments can be greatly enhanced by this means. Damping-off caused by *Pythium* has been much reduced by planting in soil with no more than 30 per cent moisture and not watering until 4-5 days after planting. Other cultural devices that aid the same end are the use of lightened, easily drained soils for seedlings, sand or sphagnum mulches over the seedbed, and improved subsoil drainage by use of gravel or tiles.

The Cotton Seedling Blight and Boll Rot Complex

It is a common experience for cotton growers to have plantings followed by poor stands, the seed failing to germinate, or the seedlings dying before or soon after they emerge from the ground. The trouble may be due to any one of several seed- and soil-borne fungi. These same organisms also may bring about boll rot and lint and seed decay later in the season.

Importance. The cotton seedling blight and boll rot complex is one of the major cotton disease problems. The losses are brought about in a variety of ways, including expense of overseeding with increased cost of thinning; cost of replanting and loss from the boll weevil and drought in the late, replanted crop; weakening and stunting of surviving plants; direct loss from boll rot; and expense of control treatments. The estimated losses range up to 15 per cent of the annual crop or 188,000 bales in North Carolina and 13 per cent or 944,000 bales in Texas with losses of 10 to 20 per cent of the crop in a number of other states, totaling a loss of a million and a half bales in 1937 and 1938 from seedling disease and boll rot. When seedling blight is controlled by seed treatment, yield increases of 10 to 30 per cent are not uncommon. Other cotton diseases are more destructive in limited areas, as *Fusarium* wilt on infested soils and Texas root rot in the Texas blacklands. But since the seedling blight and boll rot complex is

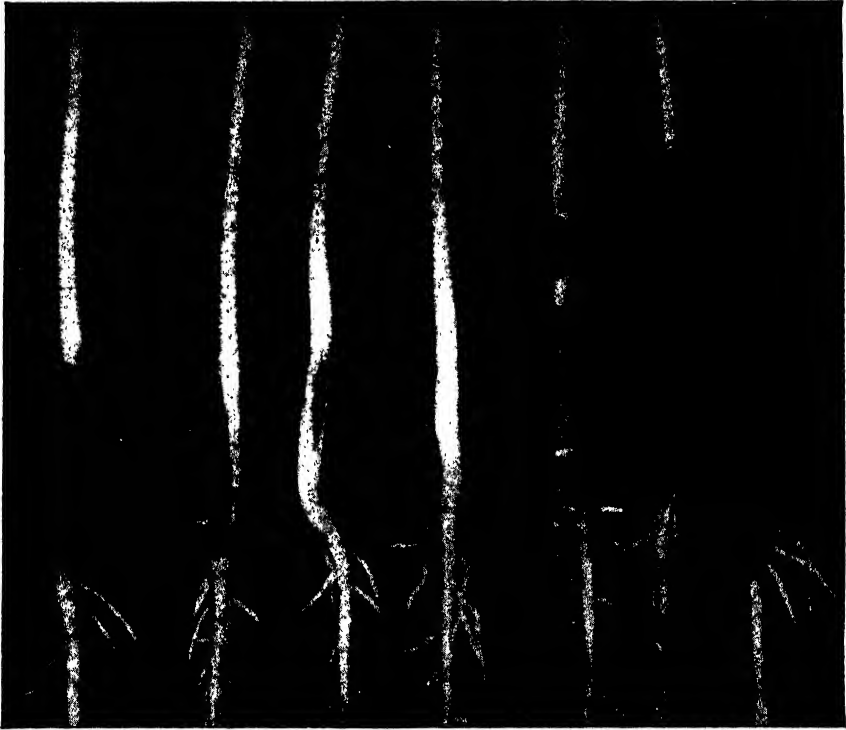


FIG. 127. Damping-off of cotton seedlings caused by *Glomerella gossypii* (left of healthy plant in the center) and *Rhizoctonia solani* (right). (Courtesy, R. Weindling, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

regularly present wherever cotton is grown, it is regarded by some as the most generally destructive disease problem with this crop.

Symptoms and Signs. Seeds infected with seedling blight organisms may fail to germinate, the seed contents being decayed to a greater or lesser extent. When placed in a germinator such seed are soon overgrown with white, pink, or dark mycelium of the pathogens. Seedlings may be attacked and killed at any stage after germination of the seed, before or after emerging from the soil. The roots and lower part of the stem of affected seedlings are decayed and shriveled (Fig. 127). Seedling blights due to various organisms attacking cotton are difficult to distinguish from one another, and usually it is necessary to culture the tissues on nutrient agar in order to determine which organism is present in any given case.

The cotton seedling disease fungi ordinarily do not cause serious injury to cotton plants through infections of the stems and leaves of older plants. Minor infections of these organs do occur, however, and these may be important in the survival and multiplication of the fungi preliminary to the boll rot phase. In the case of *Rhizoctonia solani*, one of the

leading organisms involved here, seedlings frequently survive attack and show healed-over lesions on older stems near the ground. This condition is called sore-shin, and plants so affected fail to make as vigorous growth as normal plants.

Cotton bolls show various degrees of damage from the seedling disease and boll rot fungi, ranging from discoloration to complete destruction of the lint. The different fungi cause boll rots of different appearance.

One of the principal types of boll rot is pink boll rot, caused by the cotton anthracnose fungus and shown in Fig. 128. This begins as small, dull red spots that enlarge until they may cover one fourth to one half of the boll. Growth of the boll is arrested, the lint becomes weak and discolored, and in moist weather a coating of spores on the boll surface gives the lesion a slimy pink coating. *Fusarium* boll rots have a similar appearance. Rots caused by certain other fungi (*Aspergillus niger*, *Diplodia gossypina*, *Rhizopus nigricans*) eventually give the boll a black, smutty appearance, due to production of dark spores. This condition is sometimes called "cotton smut" by growers, a misleading term, because the



FIG. 128. Cotton bolls collected from a field showing an unusual amount of anthracnose boll rot. Note the water-soaked early stages and abundant mycelium and spores covering the lesions in advanced stages. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

fungi concerned are quite unrelated to the smuts, and because cotton does have a true leaf smut disease in tropical regions.

The fate of the boll and its lint and seed are determined by the degree of decay. Often the boll is so stunted and decayed that it does not open and release the lint, and the boll is left unpicked. If there is less injury, or if not all of the locks are decayed, some lint may be picked, but this is often stained or weakened in strength. Such cotton receives a low grading and price when it is delivered to the gin.

The seed from such bolls are contaminated with spores of the boll rot fungi. When such seed are planted, if they are viable they frequently produce seedlings which are promptly attacked by the contaminating fungi, seedling disease results, and the cycle of the disease complex goes on.

Etiology. Surveys of the cotton seedling and boll diseases made throughout the cotton belt during the past decade, including many thousands of culturings, indicate that the principal organisms involved are the following:

Glomerella gossypii, the cotton anthracnose fungus, is commonly found in the imperfect stage when it is known as *Colletotrichum gossypii*, a relative of the fungi causing anthracnose diseases of beans, cereals, legumes, watermelon, and flax. Considering the cotton belt as a whole, it is the commonest cotton seedling disease fungus and the second most prevalent boll rotter. In the drier Southwest it is of less frequent occurrence. It produces pink masses of clear, one-celled, oblong conidia in acervuli. (Figs. 37, 129.) An ascospore stage is produced rarely. It is the most virulent of the seedling and boll organisms and, in the moister cotton sections, is the most important. In drier areas it yields in importance to slightly less aggressive but more prevalent fungi. It is distinctly a cotton fungus, not having a wide host range.

Next in rank is *Fusarium moniliforme*, related to the wilt *Fusarium* species. This is an imperfect fungus with a broader host range, and is involved also in the corn seedling disease and ear rot complex. It produces colorless microconidia in fragile chains, light pink in mass. In some cotton areas it is the leading pathogen of this complex because of its great prevalence. Numerous other *Fusarium* species also are found to cause cotton seedling disease and boll decays.

Alternaria species, sooty molds with dark, several-celled spores like those of the fungus causing potato and tomato early blight, are leading boll rotters (Fig. 129), not commonly associated with seedling blight. These are ever-present saprophytes with the ability, occasionally, to attack living plants. One *Alternaria* species causes a blue lint stain.

Macrophomina phaseoli, a very prevalent soil-dwelling fungus which

rarely forms spores but is recognized by black mycelium with many tiny round black sclerotia, like pepper grains, is sometimes a cause of seedling blight but, since normally it produces no spores, it cannot attack the bolls unless these are in contact with soil, in which case the mycelium can grow directly into the bolls. Many other species of plants are attacked by this frequent soil-dweller, and it plays an important part in the corn and sorghum root and stalk rot complexes to be studied later.

Rhizoctonia solani, which is already familiar as the pathogen causing potato black scurf and a leading damping-off fungus, has habits like those of *M. phaseoli*. Its coarse, jointed brown mycelium (Fig. 129) is present in many soils, it has a wide host range, and ordinarily it produces no spores. It is the principal cotton seedling disease fungus in drier areas (Fig. 127), also causing sore shin of older stems, but is not a boll rotter unless the bolls come into contact with the soil.

Rhizopus nigricans, the black bread mold fungus which was studied as the cause of soft rot of sweet potatoes and leak of strawberries (Fig. 111), is less frequently associated with seedling disease, but its spores, universally present in the air, can produce a soft boll decay of cotton.

Diplodia gossypina is an imperfect fungus in which the dark brown, two-celled conidia are produced in black, flask-shaped pycnidia (Fig. 129). These are under the surface of the epidermis of cotton, the spores exuding from the opening of the pycnidium which penetrates through the epi-

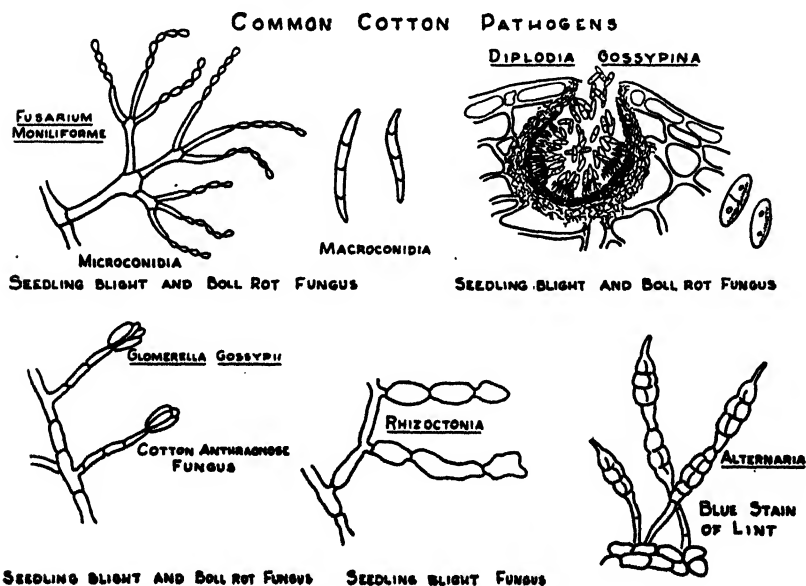


FIG. 129. Mycological characteristics of fungi that cause cotton seedling blight and boll rots.

dermis. Under some conditions masses or strings of colorless, one-celled spores are produced. The *Diplodia* boll rot, which has a smutty appearance, is less common than anthracnose and *Fusarium* boll rots.

Aspergillus niger, a common saprophyte which produces dark brown to black heads of conidia, also occasionally causes smutlike boll rot. Many other species of fungi occasionally may be found to be implicated in cotton seedling disease and boll rot or in staining lint or cotton fabrics.

With the exception of *Rhizoctonia solani* and *Macrophomina phaseoli*, all the seedling blight fungi form spores in abundance. These spores may be carried from one crop to the next in the fuzz of cottonseed from infected bolls or accidentally contaminated in picking and ginning. In many cases the seed is invaded and the organisms survive within the seed. Doubtless there is considerable survival of these fungi in the soil as well, and overwintering in the soil is a regular feature of the life histories of *Rhizoctonia* and *Macrophomina* which are less commonly seed-borne. It should be noted also that a number of the cotton organisms are "weed" fungi that occur very widely as saprophytes or fungi that have wide host ranges. None are obligate parasites of cotton. For this reason there would always be an abundance of inoculum in cotton soils even in the absence of seed infestation.

As the seed germinate and seedlings emerge, infection becomes active, the mycelium from germinated spores or from the soil invading the young roots and stems, killing many of the plants and stunting others. More or less root infection may persist throughout the life of the cotton plant. In the cases of the spore-bearing fungi, cycle after cycle of spore production will increase the disease in the field. In most cases these cycles are rather short, a matter of a few days.

The seasonal history from seedling stage to the stage of boll formation is not altogether clear in the drier states, where it is difficult to find stem and leaf infections during this period. It is quite likely that much of the infestation dies out during the hot and dry summer months, with only enough persisting in the soil to bring on new cycles of infection on the bolls during the cooler, moist fall months.

Most of the boll rot fungi are unable to enter the uninjured boll, and even the anthracnose fungus usually follows through small wounds. Bacterial boll spot infections and insect punctures appear to be the usual portals of entry of these fungi. It is quite common to find a bacterial lesion surrounding an insect puncture, and carrying a secondary infection of *Fusarium*, *Aspergillus*, or *Rhizopus*. Once having penetrated the boll, decay follows rapidly, accompanied by the production of myriads of spores which are borne by wind and insects to other bolls in successive cycles of

infection so long as weather permits. Many spores are carried to the soil where they may develop saprophytic mycelium, or overwinter without germinating, in either case serving as sources for the next year's infections.

As the cotton is being picked and ginned the partly decayed bolls contaminate the healthy seed or yield internally infected seed on or in which the fungi may overwinter.

Epiphytology. It is commonly held that seedling blight in cotton is most damaging when planting is followed by cool, wet weather. While this is true in general, some of the seedling blight organisms can be destructive under drier, warmer conditions. The anthracnose fungus attacks seedlings most actively at 77°F., with much less damage at 65° or 91°. Stem and leaf infections and the boll rot phase of this disease complex are favored by humid weather, rank growth which retains moisture within the plant frame, and conditions favoring bacterial blight and the cotton insects that initiate the boll infections.

Control. A program of seedling blight and boll rot complex control is governed by several factors: the numerous organisms involved, the difficulty in distinguishing these without laboratory tests, the interaction of several organisms in the same field, the almost universal occurrence of certain of these organisms, and the survival of the organisms in and on seed as well as in soil. Nevertheless, much has been accomplished in warding off the more destructive effects of the complex through seed treatments, crop management and, to a lesser extent, other measures.

1. **SEED TREATMENTS.** One of the early contributions to cotton seedling disease control was the discovery in 1911 that cottonseed may be delinted with concentrated sulfuric acid with a killing of all surface-borne organisms and numerous other advantages such as the possibility of better grading of seed, hastened germination, easier, more even planting, and lowered seeding rate. This method has been recommended particularly in connection with the bacterial blight of cotton but the acid treatment also destroys surface-borne fungi on the seed and thus contributes to control of the seedling blight organisms insofar as they are carried in the lint of cotton seed. Acid-treated seed, unless they are surface-treated with fungicidal solutions or dusts, carry no protection against soil-borne cotton seedling organisms. Their rapid germination reduces seedling disease, particularly that caused by *Rhizoctonia*, by shortening the period from planting to the postseedling stage, favoring escape from damping-off.

With the introduction in the early 1930's of the organic mercury dusts for seed treatment, it first became possible to launch a direct, broad, and effective campaign against seedling disease and boll rots. Coöperative cottonseed treatment tests of workers in the various cotton states have

shown that effective dusts for fuzzy cottonseed must be volatile, so that spores which could not be reached by particles of the chemical will be killed by its fumes. The organic mercury dusts, Ceresan and DuBay 1452F have been most effective and the former is most widely used. Dow 9, a volatile phenolic compound, also is promising for fuzzy seed. For delinted seed the nonvolatile seed disinfestants, such as Arasan and Spergon are satisfactory. These dusts not only kill surface-borne pathogens, but when in contact with moist soil they slowly give off a fungicide which to some extent protects the seedling against soil-borne organisms. The wide adoption of the dust treatments for cottonseed is brought out in Table 5.

In 1944 more than 25,000,000 lbs. of cottonseed were treated by commercial growers. The improved stands and yields following treatments are due to control of seed- and soil-borne disease organisms, and the phenomenal adoption of seed dusting is an indication of the effectiveness of this control measure.

Table 5

ADOPTION OF, AND PROFIT FROM, COTTONSEED TREATMENTS IN SEVERAL STATES

State	Year	Acres Planted with Dusted Seed	Percentage of Growers Dust- ing Seed	Increased Stand from Treatment	Profit from Treatment
North Carolina.....	1935	7,000	0.8	No data	Average, \$9.82 per acre
	1936	24,000	2.8	..	
	1937	200,000	23	..	
	1938	450,000	53	..	
	1939	600,000	70	..	
	1940	..	83*	..	
	1941	..	87*	..	
South Carolina.....	1935	4,000	0.3	No data	\$2,000,000 in 2 years
	1939	510,000	42	..	
	1940	815,000	67*	..	
Georgia.....	1936	20,000	1	36%	No data
	1937	80,000	4	46%	
	1938	160,000	8	41%	
	1940	985,000	51*	..	
	1941	1,156,000	60*	..	
Virginia.....	1938	8,000	70*	28%	\$11.95 per acre
	1939	19,300	60*	(yield)	
	1940	24,000	75*	..	
	1941	25,800	80*	..	
Oklahoma.....	1939	89,000	5*	36%	\$2,180,000 per year
	1942	1,430,000	80*	(4-year average)	

* Acreage planted with dusted seed.

Seed dusting has its limitations. It will bring out the best in seed but it will not bring dead seed to life nor cure internally infected seed. When clean seed is grown under the most favorable conditions, little advantage is seen from dusting. When weather is exceedingly adverse to cotton, even treated seed will suffer from seedling blight and stands may fail. With average seed under average soil and weather conditions, however, the treatments show marked success in reducing seedling disease (Fig. 130). Some growers look upon the treatment as low-cost insurance against unfavorable conditions for seedling development; to others the advantage lies in being able to plant treated seed two to three weeks earlier than normal, with consequent less risk from the boll weevil and late season drought.

The mercury dusts are not equally effective against all of the seedling blight organisms. While they are quite efficient in killing surface-borne organisms, *Rhizoctonia solani* is more refractory to the seed treatments and this is evidently an explanation why, under weather conditions particularly favorable for *Rhizoctonia*, even treated seed will suffer severely from seedling blight.

It has been seen that in general the boll rots are caused by the same organisms as cause seedling disease. A reduction in seedling disease reduces the amount of inoculum available for boll rot later in the season, hence seed treatments have an indirect effect in reducing boll rot.

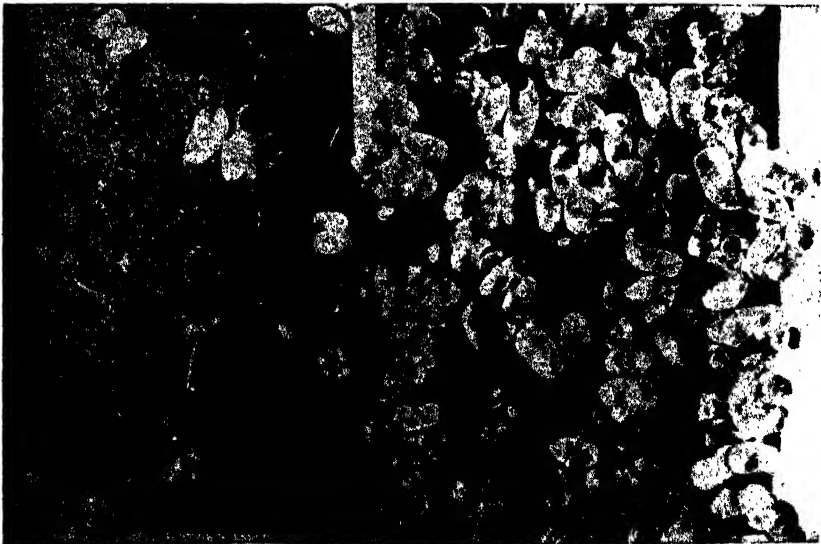


FIG. 130. Seedling blight in cotton and its control by seed treatment. Seedlings from the same number of untreated (*left*) and treated (*right*) seed planted at the same time in soil infested with seedling blight fungi.

The details of seed dusting are described in a number of experiment station publications, and accordingly only an outline of the essential points is given here. The methods of acid delinting of cottonseed are described in connection with bacterial blight on p. 278.

For fuzzy cottonseed a usual practice is to dust the seed with 1½ oz. of New Improved Ceresan. Cottonseed is often reginned to remove some of the fuzz, a practice that was compulsory during wartime to reclaim the linters which are used in explosive manufacture. If seed is not reginned excessively, it responds well to seed treatment and the amount of chemical can be reduced. Seed that is reginned so heavily that the seed coats are scratched or broken may suffer injury when treated with mercury dusts.

The dust is applied in a home-made, revolving-barrel type of treater (Fig. 19D), turned about 25 revolutions, or in a power treater such as are installed in many gins. Treatment may be carried out at any time between crops. Cost of chemical for the treatment is about 6 cents per bushel of seed. Acid-delinted seed is treated with ½ oz. of disinfestant, in either revolving-barrel or gravity type of seed treater.

The dusts are poisonous. The operator should wear a gauze dust-mask or respirator and work out of doors, on the windward side of the treater. Hands and arms should be protected from contact with the dust. Treated seed should be marked "Poison" and stored out of reach of live-stock. Cottonseed germinates well the second or third season after harvest, and if treated seed is left over after planting it should not be processed, but stored for next year's planting.

2. **RESISTANT VARIETIES.** Considering the many organisms involved in seedling and boll disease, it is not surprising that relatively little progress has been made in controlling these troubles by the use of resistant varieties. Progress is being made in development of cotton varieties that are resistant to bacterial boll spot, and since these spots are means of entry of boll-rotting fungi some control of boll rot can be expected to follow use of such varieties.

3. **USE OF OLD SEED.** The anthracnose organism dies out of cottonseed by the second season after harvest, and two-year-old seed, which germinates well, affords a simple and practical means of control of this disease so far as it is seed-borne. It is not known whether the other seed-borne organisms are controllable in this way.

4. **CULTURAL MEASURES.** Excessively early planting should be avoided, especially in tight soils. Treated seed can be safely planted two to three weeks earlier than untreated seed. In rich bottom soils care should be taken to space the plants widely enough to prevent accumulation of moist air in the dense foliage, favoring boll rot. Running over the old stalks with

a stalk cutter and turning them under thoroughly by fall plowing is helpful, because the anthracnose fungus and probably other seedling and boll pathogens live a much shorter time when buried in the soil. Under the Agricultural Adjustment Act cotton farmers are advised not to plant more than one-fourth of their acreage in cotton. This permits rotating in a two- to four-year cycle. In spite of the wide host ranges and general prevalence of the cotton seedling and boll organisms, rotation will help in decreasing the losses from these and other soil-borne cotton pathogens. Apart from the agronomic value of the practice, pathologic considerations point to the soundness of rotation as a routine practice in cotton management.

5. CLEAN SEED. Even though cottonseed is to be treated, attempts should be made to increase the effectiveness of the treatment by securing seed from fields that were relatively free from boll rot the previous year and by avoiding internally infected seed.

6. LEGAL CONTROL. One state, Oklahoma, attempts to protect cotton from anthracnose, which is seed-borne and much more prevalent in the eastern cotton states, by requiring that cottonseed for planting purposes be treated with a suitable disinfestant dust if it is to be shipped into the state.

Corn Root, Stalk, and Ear Rots

Corn, like cotton, is subject throughout its life to a complex disease problem, in this case involving root decay, stalk rot, and ear rots on the stalk and in storage. Several different fungi take part in this. The *Plant Disease Reporter* indicates very extensive losses in the United States corn crop from these rots. In the 20-year period 1918–1937, the crop suffered an average loss of 6.3 per cent or slightly more than 150 million bushels per year from this cause. About one-half of the loss was due to ear rots and about one-fourth each to stalk and root rots. These troubles are epiphytotic in certain years as, for instance, in 1926 and 1935, when nearly 60 to 70 per cent of all carloads of corn showed 5 per cent or more of kernel damage caused mainly by ear rots. In some other years the number of such cars has been much lower, with only about 3 per cent of cars in 1938 and 1939. These differences are related to seasonal weather.

The most important stalk, root, and ear rot fungi are given below, together with the essential features of their symptomatology and etiology:

Diplodia zeae is an imperfect fungus producing two-celled, olive brown conidia in black, sunken pycnidia, similar to those of *D. gossypina* seen in Fig. 129. It produces dry rot in corn ears, seedling blight, and rotting of roots and stalks. It is most common in the northern part of the

corn belt, while in the South it is replaced sometimes by a related, long-spored species, *Diplodia macrospora*. Seedlings are destroyed by *D. zeae* with a brown, dry decay. The stalks are invaded with a resulting dark brown decay of the inner tissues, often followed by lodging. The fungus often passes out of the shank into the ear where the effect varies from slight discoloration of the kernels to complete rotting of the entire ear. Pycnidia may be found late in the season on the ears or old stalks.

Gibberella zeae, the ascomycete causing scab of cereals, has been discussed (pp. 95-99). It is an ear rot fungus of leading importance. Its imperfect stage is a *Fusarium*. Other species of *Fusarium*, especially *F. moniliforme*, together with the scab organism, produce various pink ear rots, and are involved also in stalk and root rots and seedling disease. The *Fusarium* stages of all of these are distinguished by their white or bright colored mycelium freely covered with banana-shaped macroconidia and oval or cylindrical microconidia (Fig. 129). Instead of extending outward from the shank the pink ear rots usually either work downward from the ear tip or, as in *F. moniliforme*, affect individual kernels at random. Affected ears are discolored pink, reddish, or brown, and may be slightly injured or completely destroyed. The perithecia of *G. zeae* appear on the overwintered corn residue.

Basisporium gallarum is a hyphomycete with single black, globose conidia on stalks. It causes cob rot and an inconspicuous shredding of the inner stalk tissues. The shank and cob are the primary points of attack with rotting and shredding, producing dull-colored, poorly filled, chaffy ears. Spores arise from mycelium on the infected parts.

Pythium arrhenomanes, which will be discussed more fully in connection with sorghum root and stalk rots, also is damaging to corn, producing a soft root decay, especially in seedlings. It does not attack stalks and ears directly but its effects are seen in a stunting of the above ground parts. Other species of *Pythium*, soil-dwelling phycomycetes, are involved frequently in corn root and stalk decays.

Macrophomina phaseoli, which has been encountered as one of the fungi in the cotton seedling disease complex, and which will be considered in more detail as a principal organism attacking sorghum stalks, is an imperfect fungus, usually producing no spores. It invades cornstalks, eating out the pith, leaving only stringy vascular bundles covered with tiny black sclerotia. (See Fig. 132. The effect on corn stalks is very similar to that on sorghum.) In the South the disease associated with *M. phaseoli* and called "charcoal rot" often is the leading form of stalk rot.

Minor ear rots are produced by species of *Rhizopus*, *Aspergillus*, and *Penicillium*, ever-present weed fungi that are not aggressive pathogens

under field conditions but which occasionally are destructive to stored corn. They may be recognized in the crib as conspicuous green, blue, yellow, or black molds.

Although each of the stalk, root, and ear rot fungi has its own etiologic peculiarities, they may be considered together because of similarities in life histories and control measures, and because very often no attempt is made to distinguish them in grading practice.

The principal sources of inoculum are infested seed and soil. The seed from partly rotted ears may be internally infected or may be surface-laden with ear rot spores. When such seed are planted the pathogens are in a suitable position to initiate seedling infection. Likewise, the soil yielding an infested crop is likely to be heavily contaminated with spores and mycelium of the ear rot fungi, all of which can live as soil saprophytes in the debris from corn crops. Certain of the ear rot fungi initiate infection by ascospores formed in perithecia on the overwintered crop residue.

Under suitable moisture and temperature conditions, the pathogens advance into the young seedlings, producing damping-off. Spores produced on the dead parts and advancing mycelium in the soil serve for plant-to-plant spread. In injured but surviving seedlings the root infections may continue up into the stalk, producing stalk rot (Fig. 131), and thence out the shanks to the ears, or the pathogens may attack the ears directly from the tip, aided at times by earworm or corn borer injuries. During moist seasons many cycles of sporulation may occur until extensive ear decay results. When the ears are harvested, those infections that are already present may progress, and new infections may occur in the crib, especially in corn that has not been thoroughly cured and stored in a dry place.

Studies on corn root rot illustrate an important principle in these disease complexes, the attack of plants by successive waves of different fungi. Species of *Pythium* usually are the first invaders. Later these become



FIG. 131. Hill of corn killed by *Diplodia* stalk rot. The plant at the right is cut open to show rot in the pith. (Courtesy, B. Koehler, Ill. Agr. Exp. Sta.)

less prevalent and their place may be taken by *Gibberella*, species of *Fusarium*, and other moderately aggressive fungi. Finally, as the plant approaches maturity the predominant organisms in decayed roots and stalks are more likely to be soil saprophytes or fungi that are not aggressive in attack on healthy, uninjured tissues but can be very destructive after decays from preceding fungi have weakened the plant and paved the way for their attack. The charcoal rot fungus appears to act in this fashion.

Control. The essential features of a program to reduce losses from the root, stalk, and ear rot fungi include the field selection of disease-free ears, careful curing and storage of the main crop and especially of the seed ears, seed corn treatment guided or controlled by seed germination tests, rotation of corn land with early plowing of corn crop residue, and cultural practices to encourage a vigorous crop. In the extensive corn breeding programs of Iowa and other states, attention is being given to root, stalk, and ear rot resistance in inbred lines and hybrids and the newer varieties are continually showing more resistance.

Field selection of seed ears gives a much better opportunity to obtain disease-free ears than crib selection, because slight infections, resulting in a droopy shank might be unnoticed in the harvested ear. Field selection also gives opportunity to select for other desired characters at the same time: yield, smut resistance, insect resistance, drought resistance, earliness, and habit.

Drying should be at about 100°F. on racks or hangers with good ventilation, and should begin immediately after harvest and continue until the moisture content is not greater than 12 to 14 per cent. The seed ears should then be stored in a place where they will be protected from humidity, high temperatures, mice, and insects.

Germination tests should be made of 10 kernels from each of 100 ears of the seed lot. If 3 or more kernels from any ear are defective or infected, every ear should be tested, using a numbering system that identifies the kernels from each ear, and discarding all ears yielding infected kernels. A germinator consisting of a flat of wet sand covered with cloth may be used; good results are obtained also with a "rag doll" tester, made by rolling kernels in wet cloth and placing in a closed container. The good ears are then shelled separately into a sieve with hand picking of any defective kernels. The tip of the ear is broken off and discarded before shelling.

Seed treatment is most necessary when germination tests have revealed seed infestation with ear rot fungi, but is so easy and inexpensive, serves as such a good insurance against damping-off, and stimulates such vigorous growth of young plants that it is recommended as a routine practice. A proof of the popularity of corn seed treatment among farmers is seen in

the fact that 95 per cent of the seed corn for the great Iowa crop is treated each year. Nearly all hybrid seed corn is treated before sale. In general, treatments increase stands from 5 to 10 per cent, and sometimes as much as 20 per cent. This represents an addition of up to 2000 stalks per acre, a gain far out of proportion to the cost of the treatment which amounts to about 2 cents per acre. Coöperative corn seed treatment experiments carried out in many corn belt and southern states in recent years have shown good results from treating seed corn with mercury dusts (Semesan Jr., Barbak-C, etc.) or the organic dusts Spergon, Arasan, and Phygon. All are used according to the manufacturers' directions, usually at the rate of 2 oz. per bushel of seed, conveniently applied with one of the homemade seed treaters illustrated in Fig. 19. The new slurry method of corn seed treatment, which now is frequently replacing the dust treatments, is described on p. 469. Dry seed, properly treated and stored, will retain its full viability for from six months to a year. The mercury dusts are poisonous and precautions should be observed against breathing the dust or feeding treated corn to livestock.

Sorghum Root and Stalk Rots

Sorghum suffers from a root and stalk rot complex analogous to that of corn but due mainly to different organisms, perhaps because the two crops are commonly grown under quite dissimilar moisture conditions.

The first direct attack on this problem was the study in Kansas of a milo root decay known as the "milo disease," which was first noticed about 1926 in western Kansas, Oklahoma and Texas, and which proved to be very destructive in the dry-land areas. When fields become fully infested it is impossible to grow a crop of susceptible sorghums on the land. Although figures on the crop losses from this disease are not available, observers agree that it is a very destructive disease where it occurs.

The symptoms first appear when sorghum plants are 8 to 12 in. high or 30 to 35 days after planting. Growth is retarded as the leaves roll and die back from the tips. Eventually the plants die without heading or, in less severe attacks, produce dwarfed heads. There is no abnormal tillering. This blighting is due to a progressive soft, brown or red decay of the roots which may begin when the plants are no more than 3 in. high. A dark red discoloration of the tissue at the base of the crown is characteristic, but there is no primary decay of the crown or stalk (Fig. 132).

The varieties of sorghum differ greatly in their susceptibility to the disease. It is principally a disease of milo, the other sorghum types, such as the kafirs, feteritas, sorgos and broom corn, being resistant. Many of the milos are very susceptible, but selections resistant to the disease have been

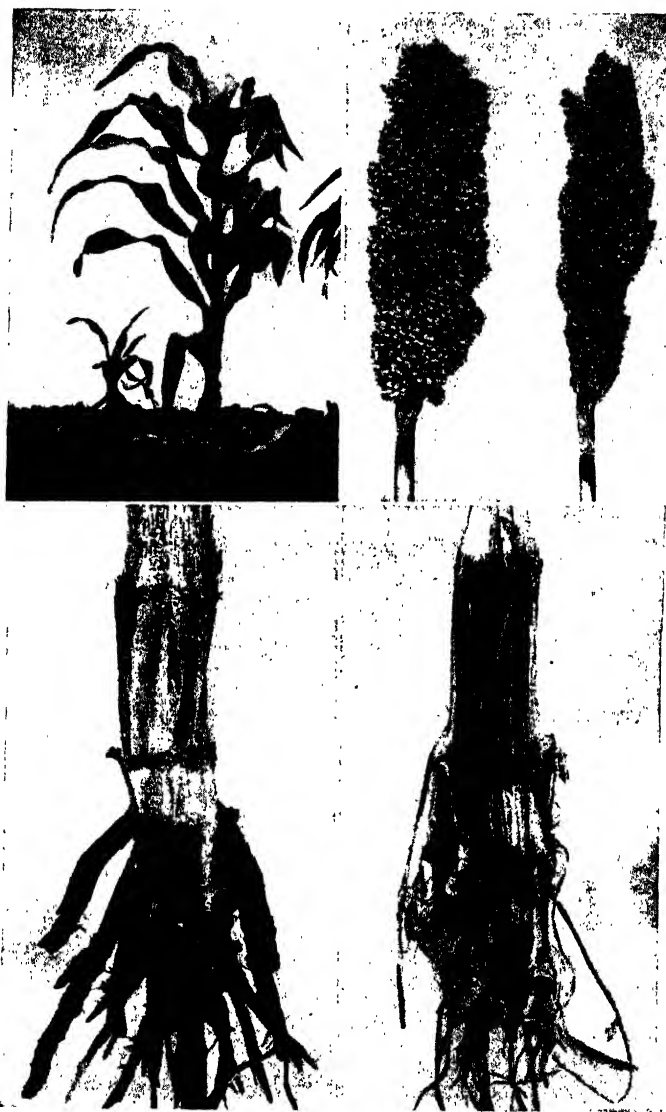


FIG. 132. Root and stalk rots of sorghum. (Left, top and bottom) Milo disease. (Left, top) Diseased and dead plants beside healthy resistant selection. (Left, bottom) Features of the stalk decay, in particular the discolored zone at the crown. (Right, top and bottom) *Macrophomina phaseoli* root and stalk rot. (Right, top) Head from a diseased plant in comparison with a normal head (left). (Right, bottom) Stringy dark stalk rot and decayed roots many of which are but papery shells.

made from many standard milos and sorghums with milo parentage. These include Finney milo, Westland, Texas Dwarf, Yellow Milo, Texas Double Dwarf Milo, Martin's Combine Milo, Resistant Beaver, and resistant selections of Sooner Milo, Darso, Day Milo, and Wheatland.

When first described, the milo disease was attributed to *Pythium ar-rhenomanes*, a soil-dwelling phycomycete which also attacks corn and sugar cane. More recently it has been learned that the etiology of the disease is complex, perhaps being due to a sequence of different fungi, including *Fusarium moniliforme* and *Rhizoctonia solani* in addition to *Pythium*, as in the corn root and stalk rot complex. The clearcut and practical results of selecting for resistance are noteworthy in view of this obscure and complex etiology.

The milo disease, whatever its cause or combination of causes, has some well-defined features. It can be seed-borne. It is a dry-land trouble, developing well at 18 to 20 per cent moisture-holding capacity of the soil. The greatest injury to sorghum plants is at temperatures between 61° and 77°F. Resistant varieties maintain their resistance over a considerable range of temperatures and soil moisture percentages. Soil amendments and fertilizers do not consistently prevent milo plants from becoming diseased when grown in infested soil.

The only satisfactory control for *Pythium* root rot, once the disease is established, is the use of resistant varieties of milo. It must be pointed out, however, that the sorghums which are resistant to the milo disease are for the most part highly susceptible to the other stalk and root rots described below, and these diseases must be clearly distinguished in a program of control by the use of resistant varieties.

During the past decade another sorghum root and stalk trouble has come to the front as a major problem in growing this crop. The disease usually is called "charcoal rot," and is associated with the imperfect fungus, *Macrophomina phaseoli*, which is found regularly in affected stalks, but this appears to be only the last and most conspicuous of a series of fungi that attack the plant in sequence, as in the case of the corn root and stalk rot complex and the milo disease.

The disease consists principally of a root rot which may attack sorghum at any stage of development. Under favorable conditions the seedlings are attacked soon after emergence. The young shoots die back as the root system rapidly decays. The stand loss may be virtually complete over rather extensive plantings. Usually, plants which survive are injured and fail to develop normally, remain stunted, or fail to head. Attacks of older stands resemble the milo disease. Progressive decay of the feeding roots produces weak plants that do not head, or in which the heads are small or

sterile (Fig. 132). The rot often advances up into the first few nodes of the stalk, causing a reddish-black pith rot, the entire central core, except for stringy remains of the conducting vessels, being rotted away. This pith decay may lead to extensive lodging, the fields resembling the effects of heavy hail. In the roots, and especially in the rotted pith may be found quantities of tiny black granules—the sclerotia or resting bodies of one of the principal parasites involved.

Macrophomina phaseoli, the outstanding pathogen in the charcoal rot complex, is a soil-borne fungus which is very prevalent in warm soils. It can attack the roots of many kinds of crop plants, including such varied hosts as sweet potatoes, corn, lima beans, cotton, cowpeas, beans, water-melons, peppers, gourds, chrysanthemums, and strawberries. Ordinarily, the fungus has no air-borne spore stage and spreads directly from plant to plant in the soil, or through such agencies as crop debris, surface water, and transported soil. The small black sclerotia are resistant resting bodies and serve to carry the fungus over unseasonable periods. On affected legume stems *M. phaseoli* forms numerous black pycnidia with spores, but these are lacking on sorghum or most other hosts of the fungus.

In sorghum and other susceptibles charcoal rot regularly affects injured and devitalized plants or plants growing under the stress of unfavorable environments, and is not a disease of vigorous plants. Various environmental factors that lead to weak host growth favor charcoal rot, including drought, excessive heat, soil deficiencies, and previous attack of other diseases and insects.

Control of charcoal rot includes cultural practices that contribute to vigorous growth of the sorghum plant and use of varieties of sorghums that have shown resistance to the disease. Desirable cultural practices include maintenance of soil fertility, tillage to conserve moisture, planting at recommended dates, rates, and depths, and control of competing weeds. Seed treatment is not specifically directed at this disease, but does favor more vigorous seedling development and this in turn gives the plants some resistance to charcoal rot. The same treatments as recommended for sorghum smut (page 59) may be used.

Many observations of sorghum variety trials in the presence of charcoal rot have shown that some varieties are regularly resistant and others highly susceptible, and that resistance to charcoal rot is inherited, providing the basis for successful breeding for control of the disease. The following sorghums have shown resistance at several stations. Kafirs: Standard, Blackhull, Weskan, Santa Fe, Rice, and Tall Red; Sorgos: Atlas, Norkan, Wild Amber, Leoti, Kansas Orange, Colman, Sourless, Sumac, and Early Sumac (Kan. Strain); African miller; Red Amber X Feterita; Tall White

Sorghum; Corneus Sorghum; and Shallu. It is of more than passing interest to note that there appears to be no correlation between resistance to this disease complex and resistance to the milo disease, since milos that are resistant to this disease have been found fully susceptible to the charcoal rot complex.

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Chapter 10

Diseases Caused by Bacteria

Bacteria are familiar causes of human diseases, such as typhoid fever, plague, tuberculosis, and numerous others. In 1881, Burrill, in Illinois, proved that fire blight of apple and pears is a bacterial disease. About 1900 Erwin Smith took up the study of bacterial diseases of plants, dedicated his life to it, and is responsible for much of our modern knowledge of these diseases. Bacterial diseases have been found affecting 150 genera of 50 families of flowering plants, and doubtless many more remain to be discovered.

Bacteriology. Bacteria parasitic on plants are rods, with or without motility. The motile bacteria have flagella which are either disposed at random over the surface of the bacterial cell (peritrichous flagella) or at one end of the rod, singly or in tufts of from two to seven (polar flagella). Plant disease is not caused by members of the families Coccaceae and Spirillaceae.

Several systems of naming the bacterial plant parasites have been used in the past. This is responsible for the fact that the corn wilt bacterium may be found under any one of five names: *Bacterium stewartii*, *Aplanobacter stewartii*, *Pseudomonas stewartii*, *Bacillus stewartii*, or *Phytomonas stewartii*. Of these, *Bacterium stewartii* is the name preferred today.

The bacteria parasitic on plants rarely form spores. They are nearly all Gram-negative, in contrast to many bacterial species that do not attack plants. Their resistance to crystal violet and related triphenylmethane dyes make these useful chemicals in isolating parasitic bacteria from bacterial mixtures, since such a dye kills most foreign organisms and leaves the plant parasites unharmed. They are identified by their appearance, growth, and temperature relations, by their development and reactions in various nutrient media and in the presence of various chemicals, and by their parasitic tendencies, which are often highly specialized to one or a few host species.

Pathology. Bacteria cause three principal types of disease in plants. In one type the disease may be necrotic, taking the form of localized killing of tissues, giving rise to blights, dead spotting, and rots. In another type of attack the bacteria invade and spread through the vascular tissues,

producing bacterial wilt diseases. In still another type the principal effect is to cause the host cells to multiply at an excessive rate, sometimes producing large masses of unorganized cells. Diseases of each of these types will be studied.

Bacteria are microscopic organisms, and the severe effects of their attack are due to the fact that they develop in plant tissues in great numbers and secrete powerful chemicals—toxins that poison and kill the host cells, enzymes that break down the most resistant plant materials, and growth-stimulating substances that lead to cancerous cell development.

In many cases the lesions of bacterial diseases become covered with an exudate of gummy or watery masses or droplets of bacterial ooze containing millions of bacteria (Figs. 134, 146). This ooze is moist in wet weather and dries down to form waxy scales during dry periods. The bacteria cannot be readily dislodged and blown about by wind, but when the ooze is softened by rain or dew, splashing or running water or insects serve to carry the bacteria to new infection courts. Wind-driven rain is a chief means of dissemination of the bacteria but even dripping dew serves to spread them from one part of a plant to another. Insects often are responsible for the spread of bacterial diseases, as in the case of the honeybee and fire blight of apple and pear, or cucumber beetles and bacterial wilt of cucurbits. Man, too, frequently is an agent of dissemination of these diseases by moving infected plants or plant parts, through tools and the handling of plants. A number of bacterial diseases also are spread on or in seed.

In order that infection may occur the bacteria must be carried into the tissues of the host plant. This occurs often by their being drawn into stomata or other natural plant openings with the drying of water droplets on leaf or stem surfaces. They may be introduced on the mouth-parts of plant-feeding insects, or on implements such as grafting knives and potato-cutting tools. In some cases wounds are normal and necessary portals of entry of the bacteria.

Within the host plant the bacteria multiply in the intercellular spaces, in vascular bundles, and in cavities formed by the disintegration of host tissues. These may be killed in advance of the bacteria, which follow after, feeding in a saprophytic fashion on the decaying remains of the cells which they have killed by their toxins.

Bacterial plant parasites can live as saprophytes on dead plant tissues, in the soil, and on laboratory culture media. Ordinarily they do not persist for long periods in the soil, being overcome by the greater saprophytic aggressiveness of other normal soil-dwelling organisms. In general, succulent plant tissues are most susceptible to bacterial attack, and tissues that

have become water-soaked are particularly favorable for bacterial invasion. Since the bacterial plant parasites do not form spores, they are less resistant to destructive agents, such as heat and disinfestant chemicals, than many bacterial pathogens of animals or saprophytic bacteria. In general, the bacterial plant pathogens are destroyed by ordinary disinfestants such as mercuric chloride, carbolic acid, alcohol, and formaldehyde.

Control. For those bacterial diseases that are disseminated by bacteria on seed surfaces, seed disinfestation is a necessary part of control. Long crop rotations usually are not needed in controlling bacterial diseases, because the bacteria normally are short-lived in the soil. One- or two-year rotations often are sufficient. Sanitation, including disinfestation of tools and destruction of infested crop refuse, in many cases is a very important control measure.

Spraying or dusting plants, which is so often of great value against fungus diseases, is rarely used for bacterial diseases. Sprays and dusts usually are applied to kill the infection threads of germinating spores before they have invaded the host tissues. Bacteria, on the other hand, are carried directly into the host tissues, past any coating of spray or dust, without passing through such a vulnerable stage as is represented by spore germ tubes on plant surfaces.

Breeding for resistance to bacterial diseases has been quite successful in a few cases but has not progressed so far as breeding for resistance to fungus diseases. This is an opportunity for productive research in the future.

Bacterial diseases vary greatly in severity from one season or location to another. Their occurrence and destructiveness is influenced profoundly by many environmental variables, among them being temperature, soil moisture, air humidity, soil nutrients, and wind. Epiphytotic development of these diseases can occur only when all of a complex group of environmental factors permits, and in the lack of one or more of these requisites we see operating a type of natural disease control so highly effective that we can look upon an epiphytotic as a relatively rare exception in nature, which does not often produce such combinations of favorable circumstances.

We find another type of natural control operating when bacterial plant pathogens find themselves in competition with organisms of the soil flora. We have seen that these bacteria do not remain alive for long periods in the soil but are crowded out or destroyed by the true soil saprophytes.

Some bacterial plant pathogens have been shown in laboratory tests to be attacked and destroyed by a dissolving substance, possibly a virus, known as bacteriophage. We know little of the role of bacteriophage in

controlling bacterial diseases in nature; most of the bacteria isolated in pure culture are resistant to bacteriophage, which suggests a tolerant relationship of long standing rather than an aggressive destructiveness of bacteriophage in nature. The practical use of bacteriophage in plant disease control is unexplored.

Bacterial Necroses

FIRE BLIGHT OF APPLE AND PEAR (*Erwinia amylovora*)

History, Distribution, and Importance. Fire blight has been known in America since the Revolutionary War. Originally an Eastern disease, it spread to the West coast about 1900 and now occurs as a destructive disease in all apple and pear growing sections of America. It has been the most important factor limiting pome fruit production in California. Annual United States losses (1930-1937) include from 1 to 9 million bushels of apples, $1\frac{1}{2}$ million bushels of pears, and considerable additional losses in nursery stock and ornamental plants.

Host Plants. Apple and pear are the most important hosts. Other members of the *Rosaceae* have been shown experimentally to be susceptible but on the majority of these the disease is rarely seen in nature. They include cherry, raspberry, blackberry, service berry, flowering quince, hawthorn, quince, loquat, strawberry, medlar, apricot, prune, plum, almond, rose, mountain ash, spiraea, and California holly.

The majority of cultivated apples and pears are susceptible, but vary considerably in degree of susceptibility. Some Asiatic pears are resistant and are being used as breeding parents to combine this resistance with suitable quality. The varieties Old Home and Orient are blight-resistant. The varieties Pineapple and Chinese Sand are highly resistant but of poor quality, although satisfactory for processing. Resistance has been reported also in the apple varieties Baldwin, Ben Davis, Delicious, Duchess, Golden Delicious, McIntosh, Northern Spy, Stayman, and Winter Banana.

Symptoms and Signs. Various parts of the tree are attacked.

1. **BLOSSOM BLIGHT**, the commonest symptom, is a killing of the blossoms, sometimes involving the entire tree, accompanied by bacterial ooze under moist conditions.

2. **LEAF BLIGHT** is a direct, partial, or complete killing of the leaves. Twig blight also is followed by secondary death of the leaves.

3. **TWIG BLIGHT** is a killing back of the new shoots in the spring and summer involving both leaves and flowers (Fig. 133). The blighted twigs often are hooked back in a characteristic fashion.



FIG. 133. Fire blight of pear. Affected shoot showing, at arrow, blackened necrotic lesion. Within a few days this entire shoot would be blackened and dead. (Photograph, Dep. Plant Pathology, Cornell Univ. Agr. Exp. Sta.)

4. FRUIT BLIGHT takes the form of sunken, necrotic cankers, gradually involving the entire green fruit, accompanied by bacterial ooze in moist weather. Secondary blighting of the young fruits may follow killing of the twigs by twig blight.

5. LIMB AND TRUNK PERENNIAL CANKERS (holdover cankers), are large sunken, necrotic lesions, maintaining the bacteria alive through summer, fall, and winter, exuding bacteria to initiate spring infections. The bacteria live in the living host tissues at the edges of the cankers.

The type of injury is determined by the season, the locality, the variety, and other factors.

Diagnosis is aided by the ready production of bacterial ooze from any of the above types of lesion under moist conditions, but ordinarily depends on the symptoms, which are not readily confused with those of any other pome-fruit disease. In doubtful cases the bacteria may be demonstrated microscopically.

Etiology. *Erwinia amylovora* is a rod, motile by peritrichous flagella. It does not form spores. It survives the winter only in the holdover cankers. In moist spring weather the bacterial slime oozes out of the cankers and is carried to the new growth by splashing or wind-driven rain or by insects (Fig. 134). Primary infections usually are in the blossoms, followed by a rapid spread to open blossoms and shoots, aided particularly by visiting honeybees. These and other insects aid in further spread. Man also is a vector, carrying the bacteria on pruning tools. Infection is followed by a

rapid necrotic breakdown of the parenchyma, with the bacteria intercellular, often in large pockets formed from disintegrated cells. In moist weather the bacteria ooze out to the surface, where further dissemination may lead to multitudes of secondary infections. The bacteria slowly work down the twigs and into the branches. With the advent of summer they become less active, remaining alive in the woody cankers formed especially at the point where a smaller branch joins a larger one.



FIG. 134. Fire blight of apple. Bacterial ooze (arrows) from an overwintered canker. Splashing rains will wash the bacteria from this ooze to young shoots, thus initiating infection. (Photograph, Dep. Plant Pathology, Cornell Univ. Agr. Exp. Sta.)

Epiphytology. Fire blight is favored by cloudy, humid weather, and by driving rains. It is most serious in highly fertile soils rich in nitrogen. Old neglected fence row or windbreak trees or old Kieffer pears around farmhouses are often reservoirs of inoculum, serving each year as the source of infection for nearby orchards.

Control. Many nostrums or worthless, patent medicine type "cures" have been advertised, but none has proved of value.

In attempts at controlling fire blight full use should be made of

recommendations available from state experiment stations or other reliable local sources. The recommendations suitable for one section or orchard are often unsuitable for another. The following are general measures to be followed in control:

1. Modify the susceptibility of the tree by avoiding excessive stimulation with nitrogen, or compensating for excessive nitrogen by application of potassium and phosphorus.
2. Follow a regular spray program for orchard insect control.
3. Avoid susceptible trees for windbreaks; take out and burn any nonbearing, neglected trees near the orchard.
4. During the regular winter pruning, cut out the holdover cankers. Repeat the pruning in the spring to detect any lesions that were missed before. Cut several inches below the lesions. Collect and burn the cuttings at once. In pruning, carry along a can of formaldehyde solution or other disinfectant, and dip or wipe the tools frequently. Paint cut surfaces with Bordeaux paint or creosote. With large cankers, shave away the dead area and paint over with Bordeaux paint or creosote. A suitable paint is made easily by thinning coaltar with creosote oil to the consistency of a thick paint.

Instead of cutting out large cankers, the cankers may be painted with a penetrating disinfectant. A zinc chloride solution is used, prepared as follows: To one quart of hot water in an enameled container add 3 oz. of concentrated hydrochloric acid and stir. In this mixture dissolve 9 lbs. of dry zinc chloride. When cool, mix with 7 pints of denatured alcohol. Store in tightly stoppered glass bottles. The material penetrates both bark and wood tissues and no bark should be removed from the cankers before treatment. Apply with a paint brush, covering the cankered area and the apparently healthy tissues for 5 or 6 in. beyond the evident margin of the canker.

5. Spraying or dusting is rarely done when fruit trees are blooming. An exceptional case is that of fire blight in apple, where a weak Bordeaux mixture spray (1-3-50 or 1-2-50) or copper lime dust (20-80) frequently is recommended.

6. Other factors considered, give preference to the more resistant varieties of apple and pear and to varieties grafted on resistant understocks.

BACTERIAL SPOT OF STONE FRUITS (*Xanthomonas pruni*)

Bacterial spot, which is prevalent east of the Rocky Mountains, is one of the most destructive diseases of stone fruits. It occurs throughout the eastern half of the United States from New York and Michigan southward. The losses, which are due to devitalization of the tree, killing of twigs, and

damage to the fruit, are sometimes very serious. In a favorable season the injury may range from 33 to 76 per cent on average trees or even 100 per cent in devitalized trees, and numberless orchards are heavily infested in such important stone fruit areas as Georgia, Illinois, and the Carolinas.

Bacterial spot produces several different types of injury in affected trees (Fig. 135). *On the leaves* the disease takes the form of numerous, brown, often angular dead spots. These dead areas later have a tendency to fall out, leaving the leaves with an appearance as though they had been riddled by buckshot. Such "shot holing" may also be brought on by other causes and is not diagnostic of bacterial spot. *On the fruits* the disease appears as numerous, small, circular brown spots, which often crack, become star-shaped, or exude a gummy flux as the fruits mature. This lowers the grade of the fruit, and makes it especially susceptible to decay. Affected trees also show cankers *on the twigs*. These are of two types. Spring cankers develop on young, succulent twigs of the previous season's growth at about the time the first leaves appear. At first they are small, dark water-soaked blisters, later drying out and becoming deep cankers which persist through the following winter. Summer cankers develop later in the summer, after the disease is well established on the leaves. At first they are water-soaked, dark lesions; later they become dark, torn, open lesions about $\frac{1}{2}$ in. long. Sometimes the cankers girdle and kill the twigs.

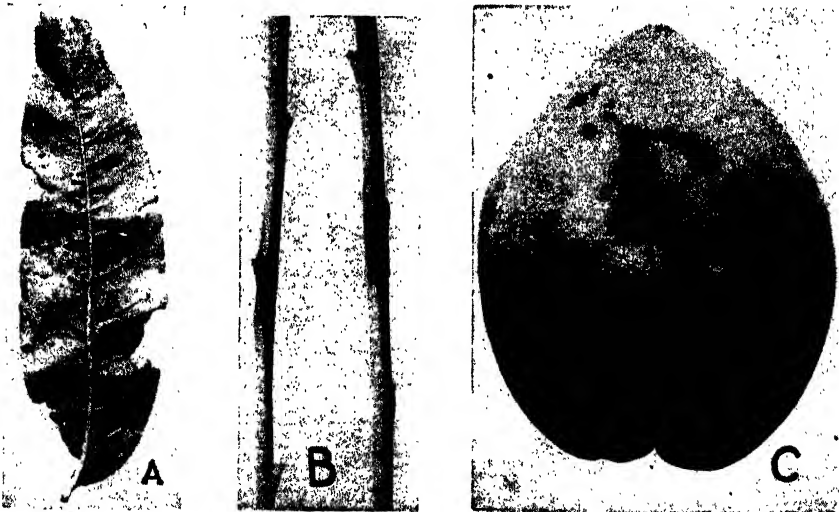


FIG. 135. Bacterial spot of stone fruits. (A) The angular lesions, at first water-soaked, later dead and dry, of spot on peach leaves. (B) Cankers on twigs. (C) Peach fruit showing several cracked lesions joined to form an extensive injury. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

The etiology of bacterial spot seems to vary in different sections of the country and in the different types of stone fruits. In Illinois, on peach, the spring infections result from bacteria oozing out of the spring cankers, which develop from infections of the preceding year that do not become evident until spring. The bacteria are spread to leaves by splashing rain or insects, and later, secondary infections develop on the twigs to form summer cankers, and on the fruits. Work in Georgia suggests that there some bacteria may overwinter in the summer cankers and initiate the spring infections. Summer cankers also are sources of spring infection in apricot and plum. In Delaware, where typical spring cankers have not been found, the bacteria overwinter in terminal buds and tissues near the twig tips. Infections of twigs and buds are responsible for wide-scale dissemination of the disease in nursery stock and budwood. Native plums and other wild or uncared-for stone fruit trees often serve as sources of infection for nearby orchards.

Bacterial spot is a disease difficult to control. Most sprays have little effect in preventing the disease, and this is a hazard also because stone fruits are highly susceptible to spray injury. Vigorous trees that are well fertilized with nitrogen do not suffer as badly as trees with less nitrogen, and it is a good plan to give affected trees an application of sodium nitrate or some other nitrogen-containing fertilizer. There is no standard spray program for bacterial spot control. Zinc sulfate-lime sprays (4-4-50) or addition of zinc sulfate and lime to sulfur fungicides used for brown rot control, are considered by some to be helpful in reducing, but not preventing bacterial spot. Others feel that the value of the zinc sulfate lies chiefly in reducing arsenic injury or in stimulating stronger growth. From Maryland there have been favorable reports of bacterial spot control by a dormant spray of Elgetol or Dinitro.

BEAN BLIGHT AND HALO BLIGHT (*Xanthomonas phaseoli* and *Pseudomonas phaseolicola*)

Beans are attacked by several species of bacteria, most prevalent of which are those causing common blight, halo blight, and bacterial wilt. All are carried from one crop to another in association with the seed. Common and halo blights are prevalent east of the Rocky Mountains, and are of utmost importance in growing beans in the South, ranking second only to mosaic in producing the heavy or complete crop losses often encountered. Similar bacterial diseases also commonly affect peas, cowpeas, soybeans, and lima beans. On the leaves the bacteria cause extensive necrotic areas, exuding bacterial ooze (Fig. 136). At high temperatures (75° to 90°F.) the two blights sometimes cannot be distinguished but under cooler



FIG. 136. Bacterial blight on bean leaves. Note the dead portions. Sometimes the injury is so extensive that growth of the plant is almost entirely stopped. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

growing conditions halo blight causes a conspicuous yellowing of the leaves, while common blight results in dead, brown areas without the yellowing. Stems are attacked with necrotic zones which in some cases encircle the stems so that they break over. Affected pods first show small water-soaked areas which enlarge, dry out, and become brick red (Fig. 137). Seeds may be completely destroyed, or may be partially infected, and thus carry the disease from one crop to the next. In addition, the bacteria can overwinter in the soil. Within the seed the bacteria may survive for many years. Spread from one plant or tissue to another is effected through the medium of splashing rain and dew, insects, and through handling the vines when wet.

Control of the three prevalent bacterial diseases of beans consists

mainly in the use of disease-free seed, preferably of blight-resistant bean varieties, and rotation of legumes. Blight is not of much importance west of the Rockies, and seed from Idaho and California is free from infection. Locally grown seed is safe only if the field was inspected and found free of the disease the previous year. The bacteria are carried under the seed



FIG. 137. Bacterial blight on bean pods, showing reddish, water-soaked, irregularly-shaped, slightly sunken spots. The seed may be invaded and serve as a source of infection for the new crop. Blighted seed should never be planted. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

coat, and ordinary surface seed treatments are not effective in control, although success has been reported from the use of very penetrating or volatile seed disinfestants consisting of bichloride of mercury in ether or aniline dyes in a mixture of alcohol and acetic acid. Particular care should be taken to avoid picking beans when the vines are wet, as this spreads the bacteria so rapidly that the crop may be quickly destroyed. Spraying is of no value in controlling blight. No commercial bean varieties are highly resistant to the bacterial blights, but some degree of resistance to common blight has been found in Refugee 1000-1, Refugee Wax, Late Stringless Refugee, and White Imperial, and to halo blight in Refugee, Scotia, New Stringless Green Pod, Black and Extra Early Valentine, Refugee 1000-1, Excelsior Wax, Logan, and Tendergreen.

COWPEA CANKER (*Xanthomonas vignicola*)

This relatively new and very destructive cowpea disease (Fig. 138) is described by Hoffmaster (see references on p. 297).

SOFT ROT OF VEGETABLES (*Erwinia carotovora*)

This is a rapid, soft, foul-smelling, wet decay of root crops, crucifers, cucurbits, solanaceous vegetables, onions, and flower bulbs. It occurs as a storage disease, and is the most frequent reason for failure of these crops to keep in storage. It may also be very destructive to cabbage, potatoes, and

other crops in the field. The bacteria are present in many soils; they enter primarily through wounds, and their destructiveness is dependent on high temperatures. Control requires thorough maturing and drying of crops before storage, storage in a cool, dry place in well aerated thin layers, avoidance of unnecessary bruising (as by pouring vegetables from one



FIG. 138. Cowpea canker. Such plants soon die, often with little or no seed production.

container to another, or throwing them into a container), and destruction of decayed vegetables to prevent the bacteria from returning to the field.

BACTERIAL CANKER, SPOT, AND SPECK OF TOMATOES

Bacterial canker (*Corynebacterium michiganense*) is a widespread and harmful seed-borne disease of tomato, destroying stems and leaves and spotting or decaying the fruit. Seedlings may be killed or stunted. Older plants suffer from a yellowish, mealy stem decay that causes the leaves to wilt and die, frequently on one side of the plant, but sometimes killing the entire plant. Cankers may appear on the stems or may be lacking. The fruit spots are first white, later cracked open and brown in the center with a white rim, often called birdseye spot (Fig. 139). Severely affected fruits are stunted, deformed, or internally infected. The bacteria are spread



FIG. 139. Bacterial diseases of tomatoes. (A) Bacterial canker, sometimes called "birdseye spot" on the fruit. (B) Bacterial spot. (C) Bacterial speck. ((A and B) Photographs, Purdue Univ. Agr. Exp. Sta. (C) Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

locally by splashing rain. When affected fruits are used for seed extraction, the seed becomes contaminated with the bacteria, and such seed has served to spread the disease widely over the United States.

Control of bacterial canker rests largely on seed treatment to destroy these seed-borne bacteria. It has been found that if seed pulp from diseased fruits is allowed to ferment for four days at a temperature near 70°F., the fermentation acids formed act as a natural seed disinfectant. The action is due to accumulation of acetic and lactic acids, and the same result can be obtained by treating unfermented seed with acetic acid. The bacteria on seed surfaces can be killed also by treating the seed for five minutes with 1:2000 mercuric chloride, followed immediately by a water wash, or with New Improved Ceresan as a dip (1:1200) or dust, but the bacteria may be internally seed-borne, in which case the surface treatments are ineffective. A hot water treatment, 25 min. at 122°F., destroys these internal bacteria

without serious loss of seed viability. Fields where bacterial canker has been damaging should not be used again for tomatoes for three, or better, four years, and the disease should be kept out of seedbeds by disinfecting their frameworks with formaldehyde and using soil that has not grown tomatoes in recent years.

Bacterial spot (*Xanthomonas vesicatoria*) is common on tomatoes and peppers in wet seasons and localities and sometimes is quite injurious. It is recognized by the presence of small, black, scabby, roundish fruit spots, sometimes with a water-soaked border (Fig. 139). The spots do not penetrate deep into the pulp, but may be the points of entry of organisms of secondary decay. On the leaves occur small circular water-soaked spots which become black and sunken. The leaves may be entirely destroyed. Elongated black lesions also occur on stems and petioles.

Bacterial speck, caused by *Bacterium tomato*, is the least damaging of these diseases. It takes the form of very small, numerous, dark brown, raised fruit spots, less than $\frac{1}{16}$ in. in diameter, not extending into the flesh of the fruit, and chiefly important by affecting the appearance of the fruit (Fig. 139). Similar spots occur on the leaves.

Surface seed disinfestation is the chief protection against bacterial spot and speck, using a mercuric chloride dip or New Improved Ceresan dip or dust treatment. If, in addition, tomatoes are grown in rotation with other crops and diseased vines are destroyed, these two diseases are not likely to give serious trouble.

TOBACCO ANGULAR LEAF SPOT AND WILDFIRE

(*Pseudomonas angulata* and *P. tabaci*)

These two necrotic leaf diseases of tobacco plant beds and rapidly growing tobacco in the field are sporadic, in some years breaking out with great destructiveness, in other years or localities being of little consequence. The diseases are described and measures for their control are given by Wolf, cited in the references at the end of this chapter.

BACTERIAL BLIGHT OF COTTON (*Xanthomonas malvacearum*)

Bacterial blight (angular leaf spot; black arm; bacterial boll spot) ranks with the seedling disease and boll rot complex as a major pathologic problem in cotton.

History and Distribution. Bacterial blight occurs wherever cotton is grown. It was first recognized in America in 1891 and the first extensive study was that of Rolfs in 1915. In the United States it is destructive from the Carolinas to the Southwest, and in irrigated areas in the western states.

In other parts of the world, as in Egypt and the African Sudan, it is a factor of great importance in cotton production.

Economic Importance. Bacterial blight is a major yield factor but since it is always present, losses often are disregarded. In the irrigated Nile Valley entire crops may be destroyed. In the Southwest up to 10 per cent of the crop is destroyed by blight. The losses are difficult to estimate because of the various ways in which the plant is affected, the disease producing poor stands, loss in vigor through destruction of leaf area, water loss through leaf lesions, stunting or killing by stem lesions, and boll decay. It is impractical to grow the long staple Sea Island cotton in the United States because of this disease.

Hosts. Cultivated cotton, and the closely related Arizona wild cotton (*Gossypium thurberi*) are the only natural hosts. Sea Island and Egyptian cottons are most susceptible; American upland cottons are intermediate and Asiatic cottons are most resistant. No commercial American cotton has a sufficient degree of resistance.

Symptoms and Signs. (Fig. 140.) *Infected seed* may fail to germinate, with the content decomposed in bacterial slime; *sprouting seed* shows a rapid yellow decay of the hypocotyl under the soil; *cotyledons* are affected with round water-soaked, then dead lesions; *leaf lesions*, often very numerous, are angular, water-soaked spots, the tissues soon dying; with many infections the leaves shrivel and fall and *young leaves* often show extensive killing along the veins; *stem lesions* (black arm) are long black cankers, partly or completely girdling the stalk; infections of *flowers* cause shed-

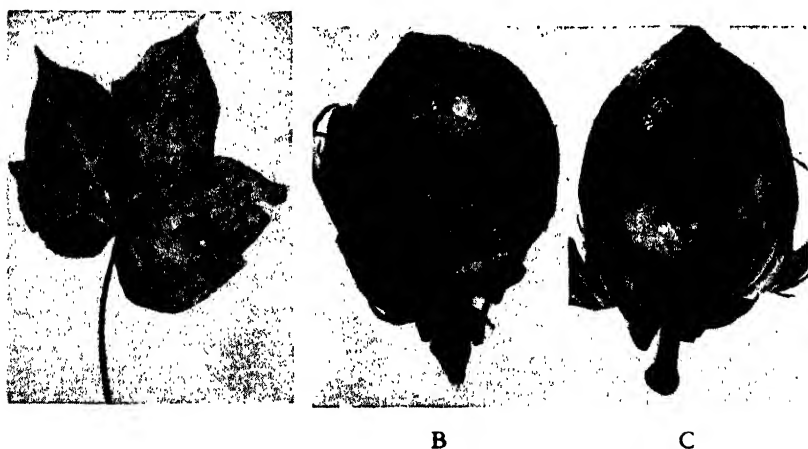


FIG. 140. Bacterial blight of cotton. (A) Angular leaf spots. (B and C) Bolls showing early water-soaked stage and advanced stage of bacterial boll spot. In the latter case secondary fungus invasion has occurred.

ding; *boll spots* are round, water-soaked, involving only the depth of the shuck, but often followed by invasion by molds which discolor or destroy the lint. In the water-soaked stage the lesions exude droplets of slimy bacterial ooze which later dries to form pale yellow scales on the lesions.

Etiology. *Xanthomonas malvacearum* is a short, motile rod, forming abundant yellow colonies in culture. It produces no spores, although gelatinous capsules occur around the cells. It overwinters mainly in the fuzz of seed from infested crops, and to a lesser extent within the seed and in the soil. As infested seed germinates, the fuzz draws across the emerging cotyledons, producing round primary lesions. These exude bacteria which are carried to later-formed leaves and rapidly spread over the plants in 10-day cycles. Eventually, lesions form on the bolls, and when seed from these is harvested, the lint is contaminated.

Further seed contamination may occur in ginning. Within the field, spread is accomplished by wind-driven rain, dew, ants and other insects crawling over the plants, and surface water. Spread from one field to another can result from wind-blown infected leaves and dust storms.

Epiphytology. *X. malvacearum* flourishes under the high temperatures (84° to 95°F.) favoring cotton growth, and is most destructive in the succulent tissues of rapidly growing plants. Hard, splashing rains produce abundant spread of the bacteria, although a high total annual rainfall is not necessarily associated with epiphytotics. Periods of cool, moist weather retard the defoliation of infected leaves, permitting the bacteria to grow down the petioles and into the stems, producing black arm. The most serious development of the disease occurs in acid soils and in soils high in nitrogen or low in phosphorus.

Control. Surface seed infestation can be counteracted by dusting the seed with organic mercury dusts or by delinting the seed with concentrated sulfuric acid. These treatments largely eliminate primary infections, substantially improving the crop but not eradicating the disease entirely, owing to internally infected seed and to volunteer seedlings from infested seed of the preceding year's crop. The few internally infected seed are not affected by the seed treatments, but are removed largely by skimming off floating seed in the wash water after acid delinting. Seed treatments coupled with a two- or three-year rotation give the best control at present available (Fig. 141). The seed treatments are effective also in destroying the anthracnose fungus and other seed-borne organisms. Delinted seed has additional advantages: saving 60 per cent of the seed by making it possible to plant with a corn plate; quicker germination and more even stands; reducing losses from skips in the row; and reducing or eliminating the cost of chop-

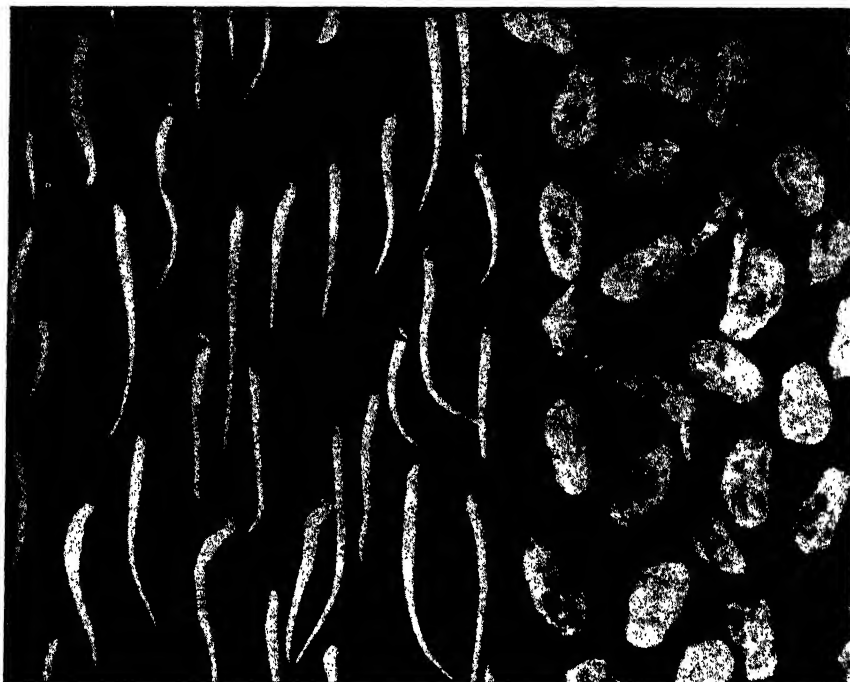


FIG. 141. When cotton seed are treated with concentrated sulfuric acid for controlling bacterial blight and other surface seed-borne diseases, the lint is removed and the seed coat becomes more permeable to water. Such seed (*left*) germinate several days earlier than similar but untreated seed (*right*). The saving in seed required for planting more than pays for the treatment.

ping. The most valuable practice of all is delinting followed by flotation grading, and then dusting the seed.

In delinting cottonseed add $\frac{1}{2}$ gal. of sulfuric acid (commercial, 66° Baumé) to 1 bushel of seed in a wooden tub. Stir until the lint is removed and the seeds form a black sticky mass. Quickly fill the tub with cold water, break up the seed mass, and pour into a second tub with a screened bottom. Rinse with several changes of water until no sour taste remains. Pour into a tight container, skim off and discard the floating seed, and spread the remaining seed out to dry. With some seed lots a very high percentage of the seed floats, in which case it is not removed. The work is simplified by a revolving barrel treater, designed in Tennessee. Details of cottonseed dusting are given on p. 252.

Despite its pronounced advantages in improving the quality and behavior of cottonseed, delinting on the farm has never been popular with farmers for a number of reasons among which rank the dangers and disagreeable features of handling strong acid, and the problem of disposing of acid wash water. Cotton seed delinting on a custom basis as a service

to growers is possible through central acid delinting plants at several points in the Cotton Belt. A high quality seed results, and the cull floating seeds, which are useful for oil crushing though inferior for planting purposes, are taken to pay for the delinting, at no further cost to the grower. In a similar way cottonseed delinting is done on a custom basis by central plants which use the Kemgas process. In this, the seed are delinted with hydrochloric acid gas and the acid remaining on the seed is neutralized with lime. Considering the reduced planting rate permissible with delinted seed it is possible for cotton growers in some areas to secure delinted, graded, and chemically treated certified seed of locally adapted varieties at a planting cost per acre no greater than the cost of planting ordinary gin-run untreated seed.

Blight-resistant cotton varieties do not offer much help in controlling the disease at present, although the very susceptible Sea Island and Egyptian varieties should be avoided. There is good prospect of control of bacterial blight in the future through use of resistant commercial varieties now being developed. The work is aided by a greenhouse inoculation method which permits large scale testing of cotton strains and selections in the seedling stage, since seedling reaction to blight is indicative of field reaction of older plants.

BACTERIAL DISEASES OF CEREALS

Halo blight of oats (*Pseudomonas coronafaciens*), bacterial blight of barley (*Xanthomonas translucens*), and three bacterial leaf diseases of sorghums—leaf spot (*Pseudomonas syringae*), streak (*Xanthomonas holcicola*), and stripe (*Pseudomonas andropogoni*)—are common diseases of these crops, sometimes resulting in such extensive leaf damage that the grain or fodder yield is seriously impaired. All are caused by bacteria which frequently ooze out of the dying leaf areas in droplets that dry to form scales or crusts. The differential symptoms are:

Halo Blight of Oats. Gray or brown leaf spots, at first oval, later streaklike with dying back of leaf tips; spots often surrounded by a pale halo; bacterial exudate absent.

Sorghum Leaf Spot. Round or oval spots, first water-soaked, then dry, parchmentlike, with a reddish border; no bacterial exudate (Fig. 142).

Sorghum Leaf Streak. Narrow, water-soaked streaks, later drying, widening here and there with oval tan centers and narrow dark margins; bacterial exudate abundant (Fig. 142).

Sorghum Leaf Stripe. Brick-red or purplish or dark brown blotches, long and rather broad; no water-soaked stage and no tan centers, but uniform red discoloration; bacterial exudate present.

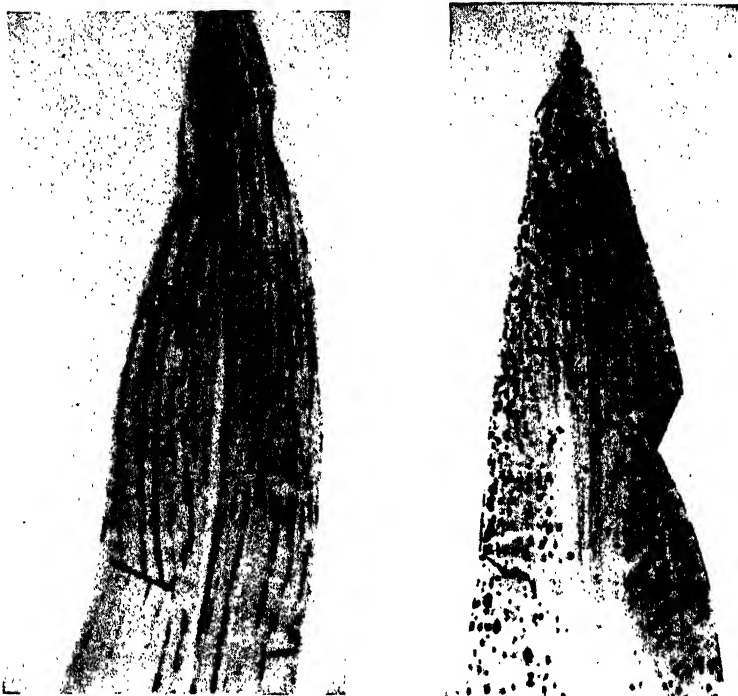


FIG. 142. Bacterial leaf diseases of sorghum. (Left) Leaf streak. (Right) Leaf spot.

Bacterial Blight of Barley. Small water-soaked, translucent spots that become elongate, then dry, causing more or less extensive killing of leaf tissues or dieback of the leaf blades; bacterial exudate present.

Etiology and Epiphytology. None of these diseases has been studied as thoroughly as its importance deserves. Each is caused by a different bacterial species but all are similar so far as present knowledge goes. Survival between crops is in or on seed or seed hulls, and in dead leaves. The plants are attacked first in the seedling stage (oats, barley) or later (sorghum) and the bacterial exudate, spread by splashing rains, dew, insects, and contact of plant parts, produces secondary infections until the greater part of the leaves may be destroyed by heading time. In all of these crops, loss of leaves in midseason definitely lowers yields. At the end of the season, bacteria carried to the heads and seed are in a favorable position to initiate next year's infections.

Control: 1. Wherever possible select seed from fields that are free of these leaf diseases.

2. Mercury dust seed treatments will protect against the inoculum which is carried on seed surfaces.

3. Few data are available on overwintering of these diseases in soil,

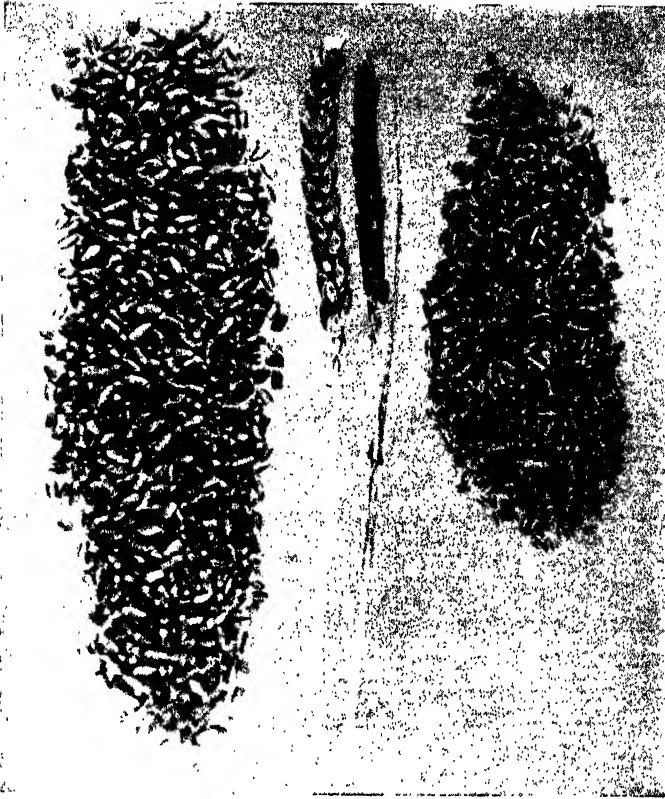


FIG. 143. Black chaff of wheat. (Left) Healthy head and grain from 50 such. (Right) Diseased head and grain from 50 similar heads.

but it would be wise to practice a two- or three-year rotation in case of serious infestation.

4. Resistance to these diseases has been observed in the following varieties:

Bacterial blight of barley: Oderbrucker, Chevalier, Reno, Woodwin, Kirwin, Wisconsin Winter, Nassau, Marm, Ward.

Sorghum leaf stripe: Sudan grass, Proso, German millet, Dwarf Early sumac, Hovey, Black Amber, Folgers Early, Early Rose, Sugar Drip, Orange, Gray Kafir, Texas Gooseneck, Guineacorn, Durra, and Shallu.

Sorghum leaf streak: a number of the kafirs, Kaoliang, Leoti Red Sorgo, Early White Milo, Buff Durra, Pierce Kafirita, and a Red Amber \times Feterita selection.

Sorghum leaf spot: no resistant varieties reported.

Oats halo blight: Clinton, some Bond hybrids, Fulghum, Gopher, Columbia, Albion.



FIG. 144. Golden Bantam sweet corn heavily infected with bacterial wilt, showing some leaves with streaks, others wilted and top wilted, and the plants stunted. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

Black Chaff of Wheat. This seed-borne disease, caused by *Xanthomonas translucens* form *undulosa*, which is fairly common and sometimes quite damaging locally, is described by Bamberg, cited in the references at the end of this chapter. (Fig. 143.)

Bacterial Wilts

BACTERIAL WILT (STEWART'S DISEASE) OF CORN (*Bacterium stewartii*)

Formerly, bacterial wilt was recognized primarily as a disease of sweet corn, especially the earlier sweeter varieties. Field corns have proved more resistant. Recently, however, the wilt problem to a large extent has been solved in sweet corn by the general adoption of Golden Cross Bantam and other wilt-resistant hybrids, while since 1932 increasing injury has been seen in field corn. Wilt is a vascular disease, i.e., the bacteria invade the conductive tissues, wilting and killing affected plants. In some years it has become epiphytotic with heavy losses.

Symptoms. Seedlings may be killed, or wilted and stunted. In larger plants, the leaves show water-soaked stripes which soon become yellow, then brown, dry out and crack (Fig. 145). The plant wilts and may die at any stage of development (Fig. 144). If the stem is cut across, the



FIG. 145. Golden Bantam sweet corn leaves showing three stages in the development of wilt leaf lesions following insect feeding injuries. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

vascular bundles appear dark and exude drops of bacterial ooze (Fig. 146). In plants which form ears the kernels may be both externally and internally infested with bacteria. Affected fields show uneven growth with many stunted or dead plants among the healthy ones. In dent corn the chief symptom is a leaf blight, in which up to 50 per cent of the leaves may be destroyed. This also makes the plants more susceptible to *Diplodia* stalk rot.

Etiology. The bacteria survive the winter primarily in the seed, or in hibernating adult flea beetles (*Chaetocnema pulicaria*) infected the previous season. The primary infections usually result from the feeding of these beetles on the new crop or as infected seedlings from infected seed, but in some cases they may come from the soil. In the field the disease is spread mainly by insects, particularly the southern corn root worm and flea beetles.

Epiphytology. The bacteria are adapted to warm temperatures, 86°F. being optimal for their growth, and the disease is most destructive in the

hotter portions of the season. Succulent plants, stimulated by rich soil and ample water, are more susceptible than plants in less favorable soil. Epiphytotics usually follow mild winters which permit extensive overwintering of infected flea beetles, and a series of such winters has a cumulative effect in increasing wilt severity. This relationship between mild winters and consequent wilt epiphytotics is so regular that it has been possible to make accurate predictions of future outbreaks, based on a study of winter temperatures, in time to adjust the planting program so as to avoid extensive losses during the epiphytotic years. Such predictions assume that the seed to be planted is not harboring the wilt pathogen.

Control: 1. Use resistant varieties. The most widely grown sweet corn, Golden Cross Bantam, is resistant, and the resistant sweet corns also include Ioana, Carmelcross 39.13, Marcross 13.6 and Whipcross C 6.2. Ordinary Golden Bantam is very susceptible. The following dent and flint corns have been reported as highly resistant: Funk 176A, Golden Glow, Golden King Yellow Dent, Iowa Hybrids 931, 939, and 942, Krug, Leaming, Murdock, Silver King White Dent, Wisconsin Hybrid (A \times hy), and Kutias. Numerous resistant dent inbreds are available for use in hybrid dent corn production.

2. Seed treatments with organic mercury dusts or bichloride of mercury are sometimes recommended though their value, except in protecting non-infested corn areas, is questionable as regards wilt. The seed treatments are not effective against bacteria within the seed and, according to recent studies, the seed is responsible for only a negligible part of spring infections in any case.

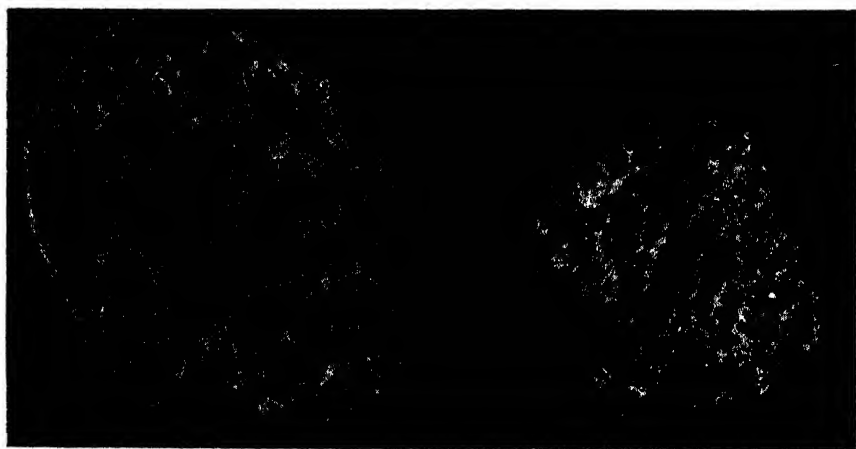


FIG. 146. Sections of stalks of Golden Bantam sweet corn showing droplets of bacterial ooze (arrows) on the cut ends of the vascular bundles. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

ALFALFA WILT (*Corynebacterium insidiosum*) •

Not very long ago alfalfa stands lasted 20 years or more before they became unprofitable. Today three- to five-year-old alfalfa fields are often so poor that they must be replanted or used for other crops. One of the causes for this is bacterial wilt, a vascular disease that has been known only since 1925. During that time the disease has become widespread in many alfalfa-growing areas of the United States where it has come to rank as the most important disease of this crop. In Pennsylvania it destroys 15 to 20 per cent of the alfalfa each year. It is destructive also in the Great Plains, Michigan, and California, but not in the eastern cotton states. Alfalfa and white sweet clover are the only susceptible crops.

Symptoms. Wilt appears in alfalfa fields the second season after planting, or later, spreading outward from spots. Attacked plants are stunted, sometimes with an excessive number of weak stems (Fig. 147). The leaves roll upward, turn yellowish, or die. Wilting is not a common symptom. The taproot, especially near the crown, shows a wet, pale yellow zone under the bark at which zone the bark easily separates from the wood. The bacteria can readily be demonstrated in the yellowed vascular tissues.

Etiology and Epiphytology. *Corynebacterium insidiosum* is a non-motile, rod-shaped organism that produces bluish colonies on lactose nutrient agar. It forms no spores. It persists in infested soil, which is the principal source of primary infection. It is considered not to be carried in seed. Infection is favored by wounds made during cultivation when the plants are wet, or by insects or winter injury. The bacteria are spread by means of soil movement, infested hay, surface water, and mowing machines. Mowing when the plants are wet is especially dangerous. The bacteria develop best at 74°F. with no growth above 82° to 88°F. The disease is most prevalent in well-watered, highly productive alfalfa, although it may be found to a destructive extent both in moist areas and in semiarid regions.

Control. The following practices are desirable:

1. Before reseeding infested fields with alfalfa, destroy all alfalfa plants and allow sufficient time for old roots to decay.
2. Sow alfalfa on fields that will not receive drainage from infested fields.
3. On land formerly infested with wilt bacteria sow winter-hardy wilt-resistant varieties. Those available and adapted to different areas include Buffalo, Ranger, resistant Kansas strains, Nemastan, Hardistan, Orestan, Cossack, and Ladak. These are only moderately resistant, but will outlive

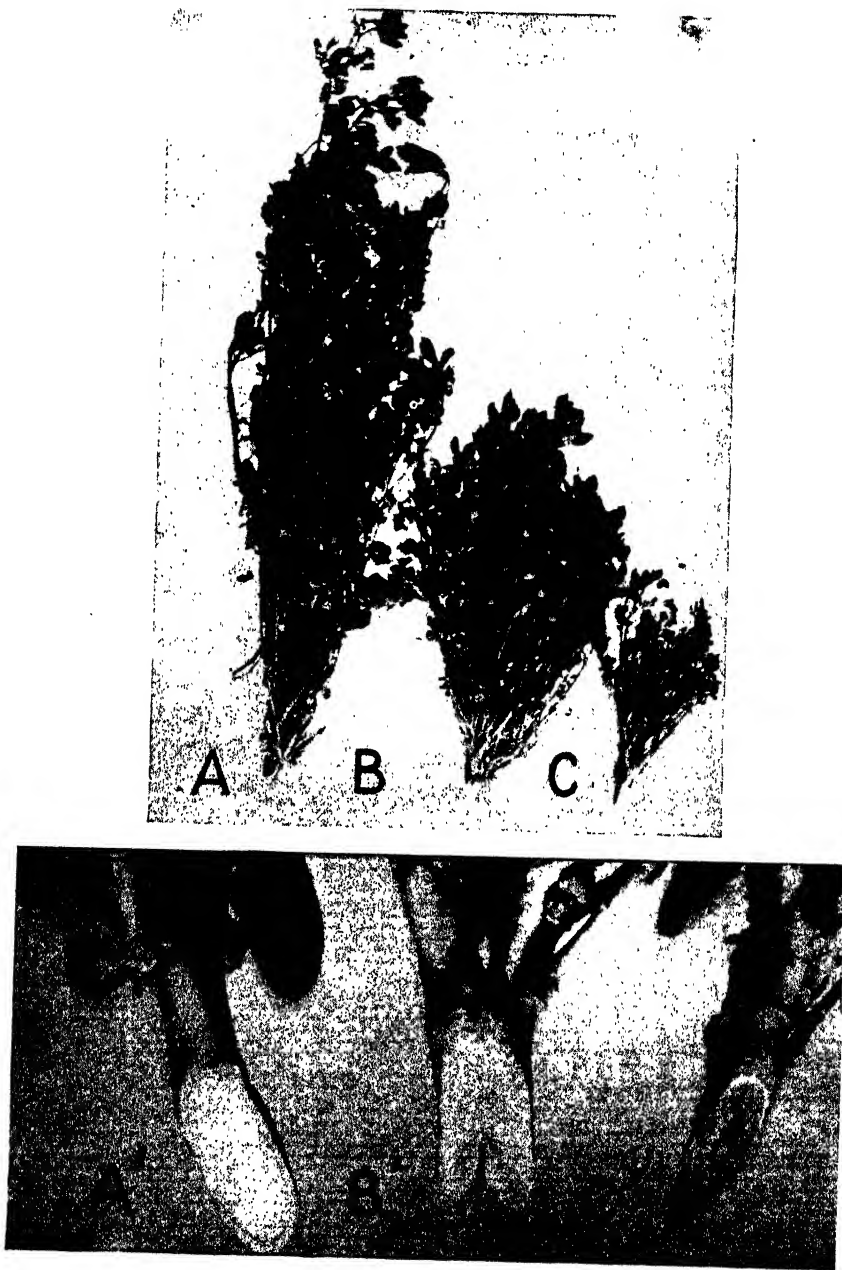


FIG. 147. Bacterial wilt of alfalfa. (A) Healthy plant. (D) Plant showing moderate wilt symptoms. (C) Plant suffering from a severe wilt attack. (A', B', and C') Shows sections of the taproots of each of these plants, bringing out the vascular discoloration that is characteristic of this disease. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

and outyield the susceptible common varieties on infested soil. They are not always superior to common varieties on wilt-free soil.

4. Cut alfalfa when the plants are dry.

5. If an infested field has been cut, wipe the mower with formaldehyde solution (1 part in 120 parts of water) before cutting a healthy field.

6. Do not topdress alfalfa with manure from animals fed on wilt-infested hay.

7. Clovers (except white sweet clover) are not susceptible to wilt and may be used in old alfalfa soils, or to fill the spots in which alfalfa plants have died from wilt. Never replant such spots with alfalfa.

8. Avoid cutting for hay or close grazing late in the season as this weakens the plants and predisposes them to winter injury, which in turn favors bacterial wilt.

BACTERIAL RING ROT OF POTATO (*Corynebacterium sepedonicum*)

This dangerous disease of potatoes has been known in North America only since 1931 when it was probably introduced from Europe, but since that time it has become widespread in Canada and the United States. The losses caused by ring rot, amounting to millions of dollars annually, are due to decay of potatoes in the field, in transit, and in storage, and to justifiable discrimination against seed potatoes containing even small amounts of ring rot.

Symptoms. Affected plants may show late season wilting, yellowing, and rolling of the leaves, but this condition is not always seen in diseased plants. The most characteristic symptoms are in the tubers three to four months after storage. The vascular ring, when invaded by the bacteria, becomes creamy yellow or light brown and crumbly or cheesy, without odor. (Fig. 148). Soon this decay involves other tissues of the tuber, and soft rot bacteria and other secondary organisms continue the destruction until the entire potato may be decayed. If affected potatoes are examined in ultraviolet light, the vascular ring shows a greenish fluorescent glow, and use is made of this in recognizing ring rot in seed potatoes.

Etiology. *Corynebacterium sepedonicum* is a small, nonmotile rod which differs from most other bacterial plant pathogens in being Gram-positive, so that in doubtful cases its staining reaction is a useful means of distinguishing it from other potato bacteria. The organisms overwinter in the vascular ring of affected tubers. When pieces of such tubers are planted, the bacteria move up into the vascular tissues of the stem, sometimes producing wilt and yellowing. As new tubers are formed they are invaded by the bacteria and serve as inoculum for the disease the following season.

There appears to be no spread from diseased to healthy plants in the

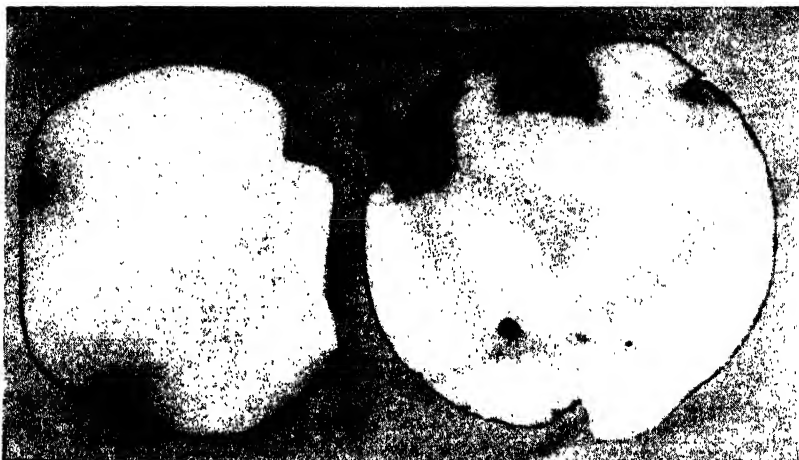


FIG. 148. Bacterial ring rot in potatoes. This odorless decay is often difficult to distinguish from *Fusarium* infection of potatoes (Fig. 71) except by microscopic and cultural methods.

field; plants from healthy seed pieces remain healthy even though growing adjacent to ring rot plants. There is no good evidence that the bacteria survive in the soil from one crop to the next. The greatest hazard lies in the fact that the bacteria can be spread from a single diseased tuber to many healthy ones by careless handling or at the time the seed pieces are cut, through bacteria smeared on the cutting knife. The picker-planter greatly increases the infestation in slightly infested seed lots, but this does not occur with hand-assist planters or hand planting. In handling potatoes the disease may be spread by contact of healthy tubers or seed pieces with diseased ones or with contaminated bins, crates, barrels, or sacks.

Control. The unusual features of ring rot—lack of field spread or survival in the soil, and spread on the cutting knife and in handling potatoes—point to effective control through use of healthy seed tubers and sanitation. Certified or well-inspected seed potatoes must be used. Most state seed potato inspection rules have very low or zero tolerances on ring rot. Table stock potatoes frequently contain ring rot and should not be used as seed. Inspection is aided by the use of the Gram stain and ultraviolet light.

Spread from an occasional infected potato to healthy ones by means of the cutting knife is avoided by sterilizing the knife. In commercial potato production, cutting should be done with a rotating knife, devised for ring rot control, which passes through a disinfectant bath of hot water or chemical between cuts. If cutting is by hand knives, several of these, resting in disinfectant solution, should be used, with frequent changing of

knives. Where seed potatoes are saved from one crop to the next it is best to maintain a separate seed plot one-tenth the size of the main acreage, planted, cultivated, and harvested before the main crop, and using disinfested barrels, sacks, knives, and equipment. Recommended disinfestants are 2 lbs. of mercuric chloride in 10 gal. water for wooden barrels and other nonmetal equipment, and 1 pint of formaldehyde in 15 gal. water, or stock dip 1-50, for metal equipment. Treatment of tubers or seed pieces with disinfestant chemicals, as for potato black scurf (p. 92), but using a weaker treatment for cut potatoes, is advised sometimes. Volunteer potato plants should not be allowed to develop, as these are the only means of survival of the disease in the field between crops.

So far no ring rot-resistant commercial potato varieties are available, but high resistance has been found in one Dutch and one British variety and in two unnamed Earline hybrids, and it is believed that it will not be difficult to introduce this resistance into commercial varieties adapted to American production.

SOUTHERN WILT (*Pseudomonas solanacearum*)

Ps. solanacearum is a pathogen primarily of the southern states where it causes wilt diseases of tomato, potato, tobacco, pepper, eggplant, and a number of other cultivated and wild suspects. Also known as brown rot or bacterial wilt, southern wilt is of limited importance in potato and tomato, but in tobacco, where it is called Granville wilt, the disease has been so destructive that tobacco was almost eliminated as a successful crop in some areas until the development of a wilt-resistant tobacco variety. The disease in tobacco occurs in North Carolina, South Carolina, Maryland, Virginia, Georgia, and Florida, and in some affected counties average losses have been 20 per cent of the crop and on individual farms as high as 90 per cent.

Symptoms. Affected potato or tomato plants show wilting with a brown, mushy decay of the stem, sometimes resulting in brown discoloration of the stem surface. The wilting becomes more and more noticeable from day to day until the plant dies. The more or less complete rotting of the stem and presence of bacterial ooze distinguish the disease from *Fusarium* or *Verticillium* wilts. Affected potato tubers first show a browning of the vascular ring, followed by general decay caused by secondary organisms. The bacteria ooze out at the eyes, and dirt adheres to this ooze, the little dirt masses at the eyes being a distinguishing characteristic of this disease. It is distinct from ring rot in the presence of slimy ooze and the dark color of the affected vascular ring. Tomatoes show no special fruit symptoms.

In tobacco, affected plants display more and more severe wilting until they die. The leaves become pale green, then yellow. The cut stalks show dark streaks with oozing bacterial slime, and the roots are more or less decayed. Whole fields may be so affected.

Etiology. On death of the plants the bacteria are liberated into the soil where they survive saprophytically between susceptible crops, or by maintaining themselves in weed hosts. In the absence of susceptibles they may be present in the soil for as much as six years, but a rotation cycle with immune crops for from three to five years between susceptible crops gives a fair degree of practical control. The disease is not seed-borne. In potato it is carried in the tubers, but this is of little importance since in the South, to which the disease is limited, northern-grown potatoes are regularly used as seed. In the field the disease is spread by irrigation water and any means of moving soil or crop debris, as on the wheels of farm implements or the hooves of livestock.

Control. In potato the varieties Green Mountain and Katahdin are resistant and may be used on infested land in place of Triumph, Cobbler, or other common varieties. Florida experiments have shown that on sandy soils bacterial wilt can be controlled by applying 800 lbs. of sulfur per acre in the summer, followed by 3000 lbs. of limestone in the fall. There are no resistant commercial tomato varieties and with this crop a four- or five-year rotation, with resistant crops and weed control in the years between tomato crops, is recommended.

In the case of tobacco there was no recourse beyond partially effective crop rotations and seedbed sanitation until the recent production of the resistant tobacco variety Oxford 26, a masterpiece of plant breeding which has restored value to abandoned, wilt-infested farms, and rejuvenated the waning tobacco culture in seriously affected counties. Oxford 26, a cross between flue-cured tobacco and a South American tobacco strain, survives from 95 to 100 per cent in wilt-infested soil in which less than 1 per cent of standard varieties survive, and shows good yields and quality whether or not the soil is infested. Previous to the release of Oxford 26, the most useful practice had been rotation of tobacco after three years of corn, soybeans, or redtop clover, or after one year of corn with a heavy application of urea.

BACTERIAL WILT OF CUCURBITS (*Erwinia tracheiphila*)

Cucurbits suffer from two types of wilt diseases, *Fusarium* wilt (as the common watermelon wilt) and bacterial wilt. The latter is widespread, somewhat more destructive in the North than in the South, but not uncommon in southern fields and in greenhouses. Cucumber is most seriously

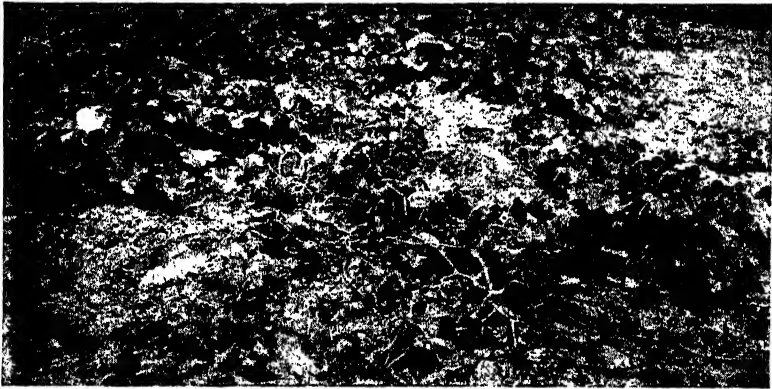


FIG. 149. Bacterial wilt of muskmelon. (Courtesy, C. T. Gregory, Ind. Agr. Extension Serv.)

attacked, followed in order by cantaloupe and squash. Watermelon is almost immune.

Symptoms. A few leaves are seen to wilt, followed soon by wilting and drying of the whole plant (Fig. 149). Cross sections of the stems show white bacterial exudate oozing from the vascular ring. Suspected plants should be examined also for *Fusarium* wilt (dark ring under the bark) and work of the squash vine borer (tunnels in the stem bases).

Etiology and Epiphytology. The bacteria overwinter in the bodies of cucumber beetles, and these are the only means of spread in the field. When introduced into susceptibles the bacteria multiply, plug the vascular bundles, dissolve cell walls and form cavities of decay in the parenchyma.

Control. Control is entirely directed at restriction of the beetles which are the agents of overwintering and dissemination. A good insecticide for this purpose is cryolite. The vines must be protected from the seedling stage till harvest begins, which may mean as many as eight applications. In the greenhouse the beetles may be controlled by fumigation. When the disease first appears, pulling of affected vines, if thoroughly done, may give adequate control.

Bacterial Hyperplasias

CROWN GALL (*Agrobacterium tumefaciens*)

Crown gall is principally a disease of young nursery trees, occurring naturally on pome fruits, stone fruits, small fruits, nut-bearing and broad-leaved shade trees, and as a minor trouble of alfalfa, cotton, some of the root vegetables, and herbaceous ornamentals. At times it is quite destructive to sugar beets. Because of a resemblance to a human disease

it has been called 'a plant tumor. It is widespread and common, and losses from crown gall often are serious in young fruit trees, not because the plants are killed, but because they are unfit for sale. It is one of the most important reasons for condemnation of nursery stock, exceeded only by root knot in warm regions.

Symptoms. In their usual form the galls are rounded, with a rough surface, ranging up to several inches in diameter, occurring chiefly at the soil line, but not uncommonly over the roots or above ground, especially at the graft union (Fig. 150). Internally, the galls show irregular structure, with the tissue elements disarranged, as in burls, at first firm and white, but later often showing secondary decay. Hard and soft galls are distinguished, the latter decaying after a season of growth. Hard galls resembling crown gall are commonly knots of graft callus or wound overgrowth. Other related types of disease include hairy root or broom root in which a large number of fine, weak roots replace the normal root system, and bur knot or wooly knot in which the gall is covered with "whiskers" of fine roots (Fig. 150). Hairy root is considered due to a species of bacterium distinct from that causing typical crown gall. Hairy root, bur knot, and wooly knot are indistinguishable in the field if the specimen is small.

Etiology and Epiphytology. Crown gall is caused by *Agrobacterium tumefaciens*, and hairy root by *A. rhizogenes*. Infection normally occurs on the grafting bench, the bacteria passing from diseased to healthy trees on

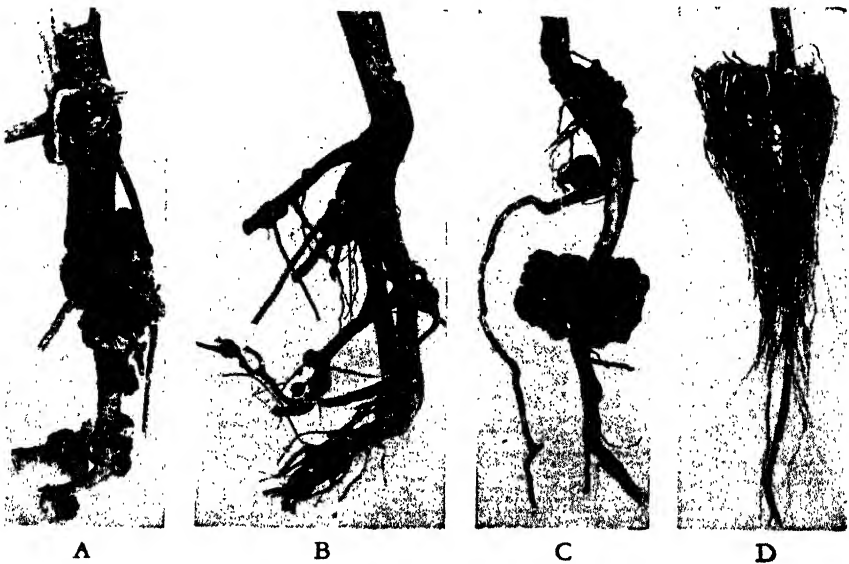


FIG. 150. (A, B, and C) Three cases of crown gall. (D) Hairy root.

the grafting tool, or are carried inside, from the surface of infested roots, as the graft is made. Moreover, many of our native soils are infested already with the crown gall organism, and healthy trees set in such soil readily contract the disease. Usually there is relatively little spread from tree to tree in the orchard but the disease can be spread down rows of trees in irrigation water. In most cases the bacteria appear to survive no more than two years in soil in the absence of susceptibles, although there is one case on record of survival for 40 years in grain land. Crown gall is likely to be more serious on limed land than on more acid land.

In contrast to the necrotic bacterial diseases, the symptoms of crown gall are rather slow in appearing, the incubation period lasting from a few weeks to a year or more, depending on weather, growth rate of the host, and other factors. Despite the extensive studies on the disease by Erwin F. Smith, there still remain many controversial or poorly understood points. One of these is the question of the amount of damage sustained by galled plants. Although trees at times may suffer severely or even be killed by crown gall and its secondary consequences, there are numerous cases of spontaneous recovery in plants not affected on the trunk, or of heavily galled trees that have continued apparently normal growth and bearing for many years.

There are a number of features of crown gall which are of fundamental scientific interest. The basic cause of pathologic growth in plants is probably not far different from that in man and other animals. Consequently, crown gall is being studied intensively in several laboratories in the hope that the results will contribute to a solution of certain problems including tumors and cancers. The production of galls is a process of stimulated, unorganized growth, somewhat analogous to the action of growth-promoting hormones. Galls closely resembling crown gall can be induced in plants by injecting them with growth-promoting substances although only at rather high concentrations. The bacteria in laboratory cultures often are associated with a bacteriophage, a dissolving principle or virus that destroys the bacteria and may possibly contribute to the natural control of the crown gall organism.

Control. To a great extent control of crown gall is a matter of sanitary practices in propagating trees and shrubs. Planting stock or propagation wood should be selected for freedom from crown gall, and the efforts of state nursery inspectors aid in this. Extreme care should be used in grafting, to avoid transmission of the disease from one plant to another. Grafts should be well fitted and bound. It is a good practice to dip graft understocks in a disinfestant solution such as bichloride of mercury (1:1000) followed by washing, copper sulfate ($\frac{1}{2}$ to 1 lb. in 26 gal. of water soaking for

1 hour) or Semesan Bel. The grafting knife should be sterilized by frequent dipping in potassium permanganate, 1 oz. in 2 gal. water, or denatured alcohol. Finished grafts should not be treated with disinfestant chemicals as this will prevent suitable decay of the graft wrapping and may lead to girdling of the tree. Infested soil should not be used for susceptible stock until a nonsusceptible crop had been grown on it for one or two intervening years. Mechanical wounds to the young trees or bushes should be avoided so far as this is possible. Other things being equal, the varieties of fruits which show some resistance to crown gall in a given locality should be given preference. Heald lists the following varieties as crown gall-resistant:

Grapes: Concord, Catawba, Delaware, and other American varieties in contrast to European varieties.

Apples: Variable in different localities; Jonathan is reported as less susceptible than Wealthy.

Stone fruits: Italian prune, German prune, Damson, cherries, especially Mazzard (for understocks for the other stone fruits).

Nut trees: California black walnut (useful as understocks for other compatible nut trees).

Miscellaneous: Loquat, silk oak, avocado, olive.

It has been repeatedly observed that varieties behaving as resistant in one locality may prove susceptible in another, and the foregoing lists must be accepted only with that reservation. In a series of hairy-root infection tests made in Kansas during two years, Hildebrand found that the apple varieties Arkansas Beauty, Early Harvest, Hopa, McIntosh, Rome Beauty, and York Imperial showed considerably less susceptibility than many other standard varieties; however, the results of one year's experiment did not always agree with those of the second year, as in the varieties Gano I (Black Ben) and Winesap which showed low infection in 1930 and very high susceptibility in 1931 experiments.

It is possible sometimes to cure galled trees by painting the galls with a disinfestant such as Elgetol plus methyl alcohol (20-80), iodine, or clove oil. Crude penicillin has been used for the same purpose. Treating peach pits with mercury disinfestants before planting has been helpful in reducing crown gall.

COMMON SCAB OF POTATO (*Streptomyces scabies* and related species)

Common scab occurs wherever potatoes are grown in the United States. It causes losses estimated at several million dollars annually, due principally to a lowering of the market grade of potatoes. Scabby potatoes

have poor customer appeal, and are wasteful because of the deep paring required. They are undesirable for seed and are more liable than healthy potatoes to decay in storage. Badly affected potatoes are culls, and usually are fed to livestock. In cases of severe scab, yields are reduced 15 or 20 per cent, by production of secondary tubers or rotting of the tubers in the soil.

Symptoms. Scab occurs only on the tubers where it forms small, brownish spots which later become enlarged to form hard, circular, or irregular corky areas on the tuber surface, or they may be cracked open, extending for some little distance down into the flesh of the tuber (Fig. 151). When they are numerous the scab lesions may run together to form

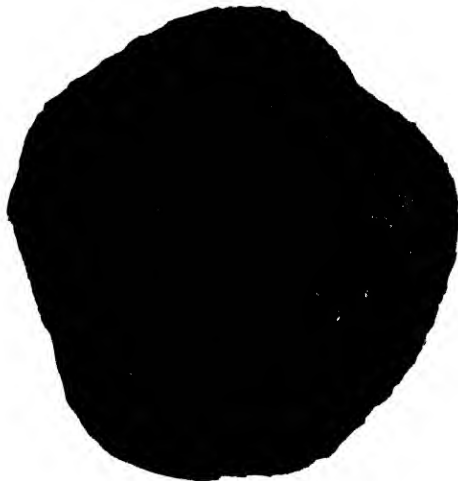


FIG. 151. Common scab of potato. Severe or deep type resulting in cracking of the tuber.

extensive, rough areas that may involve most of the tuber surface. Owing to the action of different strains of the scab organism, the reaction of different potato varieties, and the age at which the tuber is attacked, scab varies in appearance, the lesions being deep or shallow, large or small, or merely netting the skin of the tuber.

The scabbed appearance is due to abnormal production of cork cells which permits us to classify this disease among the hyperplasias, in which excessive growth is a main symptom. Some necrosis accompanies this, however.

Etiology. Common scab may be caused by any of several species of *Streptomyces* of which *S. scabies* is most common. These organisms are intermediate between bacteria and fungi. They form threadlike filaments which break up into tiny bacterial rods that germinate to form filaments

again. These can invade young tubers to produce the scab lesions. These structures can be seen as a grayish coating on freshly dug scabby potatoes. Infestations originate from the planting of scabby tubers or from the organism in the soil. Even virgin soils sometimes contain it. Sugar beets and a number of other fleshy-rooted crops are susceptible. Potato varieties differ in scab susceptibility, and the varieties Russet Rural and other Russet types, Sebago, Menominee, and Katahdin range from moderately to very resistant.

Epiphytology. This is a classic case of a disease that is highly influenced by soil reaction. It is most destructive at soil pH 5.7 and very little infection occurs in slightly more acid soil, at pH 5.2 to 5.4, which makes it possible to control the disease, in some cases, by acidifying the soil with sulfur. The scab organism can withstand extremes of temperature and moisture but develops most abundantly at fairly high soil temperatures (between 72° and 86°F.) and low soil moisture. The organism passes unharmed through the digestive tract of livestock and may be carried to the field in manure from animals that have fed on raw scabby potatoes.

Control. The facts given above point to control of potato scab through use of disease-free seed tubers planted in scab-free soil, disinfestation of seed tubers, acidification of the soil, farm sanitation, and use of the more resistant potato varieties on infested soil. Potato seed tuber inspection removes the worst infected tubers from the seed trade, but the disease is so common that seed potatoes often show limited amounts of scab. Such potatoes should be treated before planting. In most states the preferred treatment is with unheated formaldehyde, 1 pint of formalin in 30 gal. of water, in which the uncut tubers are soaked for 2 hours. In some states, but not in all, favorable results have been obtained also by treating seed with wettable sulfur, Tersan, and Fermate. Mercury treatment often fails to control scab and may even increase it.

Where soil has produced a scabby crop it may be desirable to use a three- or four-year rotation with nonsusceptible crops. Acidification of the soil with sulfur, from 300 to 2000 lbs. per acre, is quite effective in scab control in areas of somewhat acid soils, notably the Atlantic Coastal States, but less so in many other sections where soils normally are nearly neutral. Amendments of materials that give soil a more alkaline reaction (lime, wood ashes, manure) are likely to increase the damage from scab. The scab-resistant potato varieties offer a solution to the problem where soils are infested and sulfur amendments are incompletely effective in control.

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Chapter 11

Diseases Caused by Viruses

Significance of Virus Diseases

Among the most interesting and elusive of plant pathogens are the viruses, mysterious entities too small to be seen with the light microscope or to be retained by bacteria-holding filters, yet contagious and transmissible from one host organism to another, and capable of causing some of the most puzzling and destructive diseases not only of plants but also of animals and man.

The list of virus diseases is an imposing one. It includes infantile paralysis, sleeping sickness, smallpox, typhus and trench and yellow fevers, a whole group of children's diseases, the common cold and even warts. In domesticated and wild animals viruses cause, among other diseases, foot and mouth disease, infectious anemia, swine fever, rabies, distemper, and sleeping sickness of horses, which in a single season wiped out one-third of all the horses in an epizootic area. It is not improbable that certain forms of human cancer also may be caused by viruses.

Best known of the plant virus diseases is the group of mosaics in which normally green leaves become variegated with yellow. Of these, one of the more notorious is tobacco mosaic, on the average the most destructive disease of tobacco in the North Carolina area and elsewhere. In 18 years the production of sugar in Louisiana dropped from 400,000 tons to about 50,000 tons, as sugar cane mosaic gained a throttling grip on the industry, although the adoption of mosaic-resistant cane varieties now has raised production nearly back to the former high level. Leguminous crops are subject to many mosaic viruses that regularly devitalize the plants, cutting seed and hay production by an average of 30 per cent and sometimes much more, as in the case of fall-planted beans in the South which are often a total loss when following a mosaic-infected spring crop. Farmers in the Corn Belt have come to know the destructiveness of wheat mosaic, and orchardists and nurserymen in the Southwest have suffered destruction of thousands of their trees in order to protect healthy orchards from peach mosaic.

Yellows, a second form of virus disease, has played havoc in vegetable and ornamental plantings and forced some growers of stone fruits out of

production. Still another type, curly-top, has proven to be the ranking problem in sugar beet production in the United States. In 1934, 88 per cent of the sugar beet acreage in the Twin Falls area of Idaho—18,000 acres—was abandoned because of the almost total destructiveness of the virus disease curly-top, and similar losses occurred throughout the beet-growing sections of the western states.

These viruses stunt the growth of plants and reduce their yields, but another group of viruses embraces killers. Scores of thousands of highly prized elm trees have fallen victim to the phloem necrosis virus in its rapid spread across the central United States. Spotted wilt has devastated tomato plantings, quickly destroying the vines, and in a slow, wasting decline, psorosis or scaly bark has led to destruction of many citrus trees in Florida and California.

A number of interesting and important viruses attack ornamental plants. At times these diseases produce a variegation in the leaves that is esteemed as enhancing the value of the ornamental plants. Among tulips, for example, "broken color" of the blossoms (variegation) is regularly due to viruses, and such varieties as "Sensation" and "Rembrandt" owe their distinctiveness to virus infection (Fig. 152). Indeed, the tulip variety "Farncombe Rembrandt" is merely the ordinary Rembrandt with two vicious virus diseases, perpetuated in such cases by vegetative propagation.



FIG. 152. "Breaking," a virus disease of tulips, often enhances the beauty of the flowers. (*Right*) Naturally occurring "broken" or mosaic Clara Butt tulip. (*Left*) Healthy blossom of the same variety. (*Center*) Another type of breaking obtained by inoculating the plant with cucumber mosaic virus. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)



FIG. 153. Cane fruit mosaic, showing various types of mottling and distortion on raspberry leaves. (Photograph, N. Y. (Geneva) Agr. Exp. Sta.)

Unfortunately, the varieties that are ornamentally diseased may serve as sources of infection for other varieties in which the effects of virus disease are not at all desirable, so that we find undesired virus diseases playing an important part in the pathology of most of the vegetatively-propagated ornamentals.

Even bacteria suffer from virus disease, here called bacteriophage, due to an infectious principle that spreads through bacterial colonies dissolving the individual bacterial cells.

The wide host distribution and the scientific and practical importance of virus diseases of plants are indicated by the fact that some 5,000 papers and a few books had been written on the subject by 1935, these papers dealing with virus diseases in more than 1,000 species of plants distributed through 450 genera of 90 families in the plant kingdom.

Symptoms of Virus Diseases

A great variety of symptoms is seen in the various virus diseases. When introduced into a number of different hosts a single virus may produce a range of symptoms varying from none (masked or symptomless condition) through a variety of more or less striking reactions up to extensive killing of tissues or entire plants. Or if individuals of a single host species such as tobacco are inoculated with the different viruses to which this plant is susceptible, each different virus produces a distinctive and characteristic reaction in the plant. Virus symptoms of all types show a tendency to be most evident under cool growing conditions, and to be less apparent or entirely masked at high temperatures. The leading types of virus symptoms are:

Inhibition of Pigment Formation: CHLOROPHYLL. In the *mosaic* diseases the chlorophyll in leaves formed subsequent to inoculation is suppressed in certain leaf areas. These areas usually are patches scattered over the leaves in a random fashion (Figs. 153, 160, 161). When a leaf shows white or yellow areas in regular, geometrical designs, as large V-shaped areas between the veins, or only along the borders, the variegation is more likely due to a genetic defect than to a virus, while if the patterns are symmetrical with respect to veins and leaf tips and margins, a mineral deficiency, root decay, or other interference with nutrition or environmental cause may be responsible for the yellowing. (Figs. 188, 195.) In parallel-veined plants the mosaic usually occurs as broken yellow streaks (Fig. 162). In *ring spot* which is almost always due to viruses, the variegation has the form of yellowish rings against a green background. *Clearing of veins*, usually the first virus symptom seen after inoculation, is a transient pattern in which the veins are light against a dark background (Fig. 154). The reverse of this condition, dark veins against a lighter background, sometimes occurs, in which case it is called *vein banding*. In *yellow*s diseases, entire leaves are uniformly yellowed (Fig. 155). The mosaic and ring patterns are distinguished easily from chlorosis

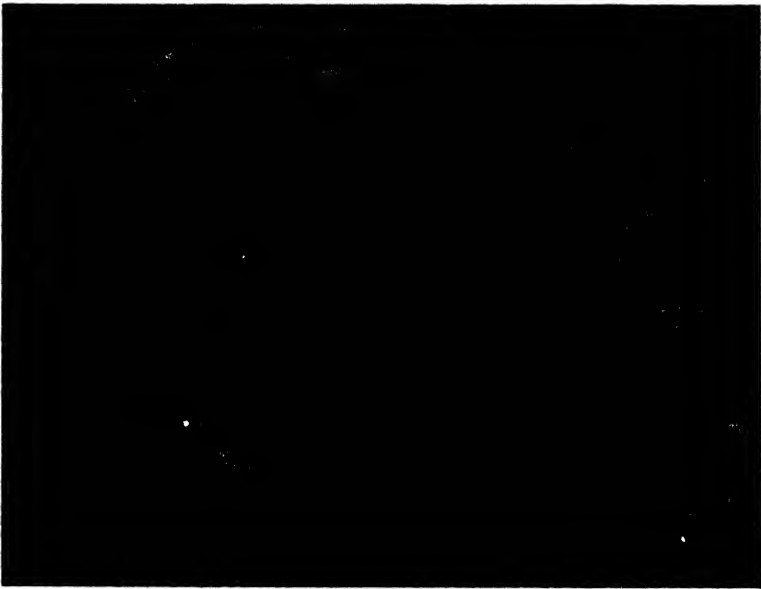


FIG. 154. Virus disease symptoms in cowpea and Jimson weed. (*Left*) Distortion in compound leaves and clearing of veins in cotyledonary leaves of cowpea infected with cowpea mosaic. (*Right*) Clearing of veins in lower left cotyledonary leaf and vein-banding in the uppermost leaf of Jimson weed infected with latent mosaic virus of potato.

due to unfavorable soil or growing conditions, as the latter is either uniform over the leaf, or begins at the leaf tips and margins and moves inward.

ANTHOCYANIN and other blossom pigments may be inhibited as in the case of chlorophyll, giving the red-and-white and other variegations seen in many ornamental plants (Fig. 152).



FIG. 155. Peach yellows. The upright habit of growth, abnormal number of shoots, slender branches, and small narrow leaves are typical of the disease. (Courtesy, L. O. Kunkel, Rockefeller Inst. for Med. Res.)

Reduced Growth. As a consequence of failure in chlorophyll formation, virus-diseased plants usually are reduced in size, 30 per cent or more reduction being not uncommon (Fig. 163). This takes the form of shorter internodes, smaller leaves and blossoms, and results in reduced yields.

Distortions. Leaves and flowers of affected plants are not only discolored, but often very irregular in shape, twisted or contorted, abnormally narrowed (strap-shaped leaves), puckered, or covered with raised, blister-like areas due to greater growth of the green areas than of the chlorotic ones (Figs. 153, 154, 163, 167).

Hypertrophy. Hypertrophy or excessive growth sometimes is a characteristic of virus diseases. It is seen in the excessive branching that characterizes such diseases as *witches' broom* of potatoes and in the production of little finlike outgrowths (*enations*) as in certain cases of tobacco and pea mosaic.

Abnormal Growth Form. The habit of the plant may be markedly changed by virus disease. Examples are the rosette diseases of peanut, peach,

and some other plants, in which the internodes are greatly shortened to produce a *rosette* habit in place of the normally elongated one, and *spindle tuber* of potato where the tops are abnormally stiff and erect, the leaves pointed upward, the tubers resembling sweet potatoes in shape (Figs. 155, 164, 168).

Necrosis. Necrosis or killing of tissues due to viruses may be either local or general. Roundish dead spots are produced on the leaves of certain plants (*local lesions*) when inoculated with viruses that do not become systemic (Figs. 157, 159). In other cases the necrosis takes the form of dead rings with green centers (*necrotic ring spot*, Fig. 158), *streaks* on stems, or an oakleaf-like outline within the leaf (*oakleaf pattern*). This last may be either chlorotic or necrotic. In severe cases the necrosis may become general, spreading to the growing point which is killed, soon resulting in death of the entire plant.

Combinations of Symptoms. Usually when a plant is infected with a virus disease it displays a combination of several of the symptoms described; for example, it may first show vein clearing, followed by a general mosaic pattern, resulting in stunting, often accompanied by some form of distortion, and perhaps eventually exhibiting local or general necrosis.

Internal Symptoms. While viruses cannot be seen with the ordinary microscope, examination of infected cells often brings out characteristic virus disease symptoms. In addition to loss of pigments, reduced size of cells and cell elements, and occasional necrosis, certain of the viruses produce foreign bodies in the cells known as *X-bodies*. These are crystalline, fibrous, or irregular in shape. It cannot be said whether they represent masses of virus or unusual by-products of the host cell.

Properties of Viruses

Before the invention of the electron microscope, when viruses could not be seen, modern science found means of determining their properties: they could be measured, counted, weighed, and many of their other characteristics could be determined.

Infectiousness. If a virus is transferred from one susceptible plant to another by suitable means, the second plant becomes infected, shows symptoms of disease, and in turn is able to infect other plants. In studying a new suspected virus disease it must first be proved that the disease is infectious. Viruses differ in their degree of infectiousness. Tobacco mosaic virus is an extreme case in that the juice from a mosaic-diseased tobacco plant can be diluted with 1,000,000 parts of water, or one drop of virus juice in a barrel of water, and the diluted juice will still infect tobacco plants if rubbed on the leaves. Other viruses cannot be diluted to this extent and some cannot be diluted at all and remain infectious.

Obligate Parasitism. All viruses, so far as is known, are dependent on living cells for their growth and reproduction; no virus multiplication

occurs in lifeless media, although a virus, like a fungus spore, may retain inactive viability for considerable periods apart from the host. But we know viruses only for their effects in producing disease. The world about us might be teeming with saprophytic viruses that we do not recognize because they cannot be seen and because they fail to produce some detectable effect, such as disease, by which we might recognize them.

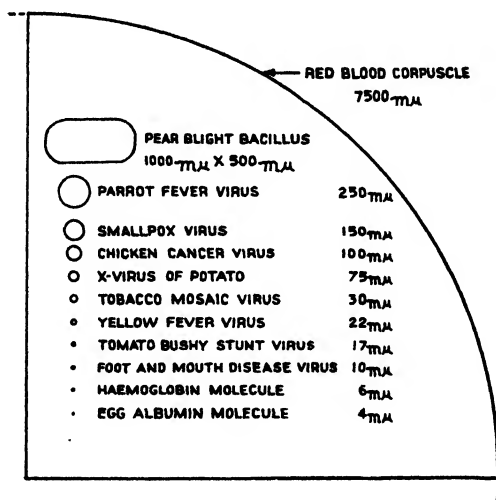


FIG. 156. Scale illustrating the relative sizes of viruses and certain other familiar types of small bodies. Shapes of the particles have not been considered, although it is known that some of these bodies are not spherical. The same relative proportions are seen if the blood corpuscle is compared to a $7\frac{1}{2}$ ft. cable spool as used for power and telephone lines, the pear blight bacillus to a football, tobacco mosaic virus to an English pea, and the molecule of egg albumin to a turnip or radish seed.

Size and Shape. Viruses may be measured by several means one of the commonest of which is to force virus-containing plant juice through collodion filters of known pore size, which can be determined by physical means. If a series of these ultrafilters of graded pore sizes are used, the virus size can be estimated by noting the smallest pore size through which infectious juice can be drawn. While this method is subject to some errors (on account of virus particle shape or adsorption of particles to pore walls), it agrees fairly well with other methods of virus measurement in indicating that most viruses are from $\frac{1}{10}$ to $\frac{1}{1000}$ of a micron in diameter. The micron (μ), $\frac{1}{1000}$ millimeter, is the unit commonly used in measuring fungus spores and bacteria. The unit used in measuring viruses is $\frac{1}{1000}$

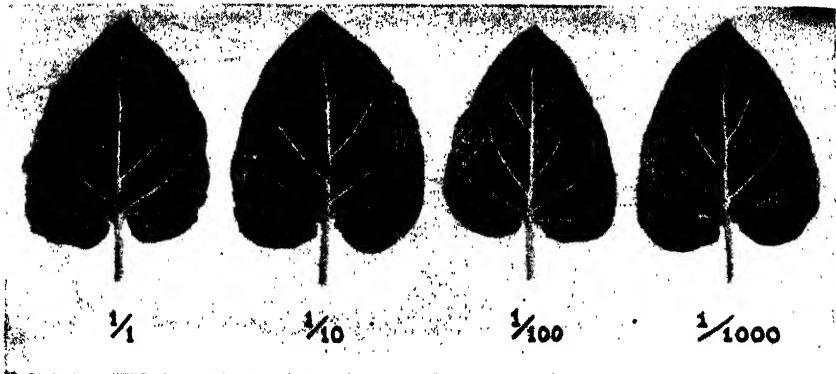


FIG. 157. Local necrotic lesions of tobacco mosaic on *Nicotiana glutinosa*. The figures give the dilutions of virus-containing tobacco juice, and the numbers of lesions shows how virus concentration may be estimated by "plating out" juice on leaves of this plant. (Courtesy, F. O. Holmes, Rockefeller Inst. for Med. Res.)

of a micron (1 millimicron = 1μ), and the virus sizes given above are thus 1 to 100μ . The resolving power of the ordinary microscope is about 0.5μ or 500μ , and the smaller bacteria are about $1 \mu \times 0.5 \mu$ in size, or 10 times to 100 times the size of the viruses. (Fig. 156.)

By using optical properties and other indirect methods, it has been determined that viruses differ in shape, including long and short rods and spherical bodies. Invention of the electron microscope first made it possible to verify this. At magnifications of 10,000 to 20,000 or higher a number of viruses have been examined with the electron microscope and found to be of several forms, including rods, spherical coccuslike forms, and forms in which several coccuslike particles are grouped in regular clusters, as in the microorganism *Sarcina*.

Weight. The weight of virus particles is determined, among other ways, by precipitating them in a high speed supercentrifuge. Knowing the rate of sedimentation and the centrifugal force, it is possible to determine the particle weight, and this has been done for several viruses, the results usually being of the order of several millions (in terms of molecular weights). There is some question, however, whether the determinations refer to individual virus particles or to clusters or aggregates of virus units.

Number. Bacteria can be counted by spreading them in a dilute layer over the surface of an agar plate and counting the bacterial colonies that develop on the plate. Viruses in some cases can be counted in a similar fashion. When the virus of tobacco mosaic is smeared on leaves of bean or of the wild tobacco, *Nicotiana glutinosa*, a little round local necrotic lesion appears in a few days at the site of each infection (Figs. 157, 159). The number of lesions bears a definite relation to the number of virus particles

in the juice smeared on the leaf, and while this does not give us the absolute number of virus particles in the juice, it does give a means of titrating unknown virus concentrations against standard concentrations. Much of the knowledge we have on the properties and nature of viruses depends on the use of the local lesion method of counting viruses.

Transmission: BY MECHANICAL MEANS. The more highly infectious viruses such as those of tobacco, tomato, and cucumber mosaics, are readily transmitted by rubbing the juice of an infected plant onto the leaves of a healthy one. So contagious are these viruses that merely to rub the fingers on a diseased leaf or to handle smoking tobacco (which commonly contains mosaic virus) and then to rub a healthy plant is sufficient to produce disease in the latter. In practice, handling plants during transplanting and pruning is a common means of virus spread. In experimental work, these viruses sometimes are transferred by pricking the leaves with needles wet with virus juice, by rubbing them with virus wet gauze or by spraying them with virus juice in a forcible stream.

BY INSECTS. The majority of viruses are spread in nature by insects. In some cases the insect merely acts as the wet needle mentioned above: it feeds on a diseased plant, its proboscis is wet with the virus juice, and in its feeding on the healthy plant some of the virus is transmitted to the latter mechanically.

But in many instances the insect is the only natural means by which the virus can pass from one plant to another. Here there is an obligate relationship between virus and insect. The insect feeds on a diseased plant, the virus passes into its digestive tract, and only after an incubation period of several days or weeks does the insect begin to transmit the virus to healthy plants. Thereafter throughout its life it continues to transmit the virus to every plant on which it feeds. During the incubation period the virus appears to multiply in the insect's body, it passes about until it reaches the salivary glands, from which point virus is supplied to healthy plants in the process of regurgitating saliva during feeding.

The virus:insect relationship often is very specific with those viruses that are transmitted in nature only by insects. The vectors involved usually belong to groups with sucking mouth parts, especially Homoptera (aphids, leaf hoppers, plant hoppers, white flies). But not just any insect with sucking mouth parts will suffice. Of all the insects feeding on peach trees, only the plum leaf hopper, *Macropsis trimaculata*, transmits peach yellows. Forty-eight other insects, including the most common sucking insects on peach, have been shown to be unable to transmit this virus which is transmissible in no other way except by grafting.

In other cases the relationship between virus and insect may not be so specific, especially when the insect acts only as a mechanical, not a biological, agent of transmission.

BY VEGETATIVE PROPAGATION. Most viruses are not carried in the true seed from infected plants, but they pervade all other parts of the plant and are transmitted by any type of vegetative propagation. Grafting and budding are common means of spreading infection in woody plants, and are useful ways of experimentally transmitting those viruses that cannot be spread by mechanical means. Bark grafting is a useful way of transmitting the scaly bark virus of citrus. Natural root grafting sometimes spreads viruses from one tree to adjacent ones, as in the scaly bark disease, and the phloem necrosis of elm. When a plant is normally propagated by vegetative parts, as in potatoes, sugar cane, bulbous plants, and ornamentals grown from cuttings, every vegetative descendant of a single virus-infected plant will in turn be infected. This perhaps accounts for the particular importance of virus diseases in the plants that are vegetatively propagated.

BY SEED. Though few viruses are transmitted in the true seed, some of the legume viruses, notably of the mosaics of bean and cowpea, are carried in this manner, and a few other cases outside the legume family have been noted.

BY SOIL. Very few viruses are transmitted in soil. An exception is wheat mosaic which regularly survives in soil and affects subsequent crops.

BY DODDER. Dodder (see p. 340), a parasitic vine, sometimes attaches its stems to two nearby plants, forming a living bridge. Across the bridge viruses may pass from diseased to virus-free plants. This method of transmission has been of great use in virus study, making it possible to transmit viruses to plants in which they can be better studied, when there is no other way of infecting the plants, because insect vectors of the natural host will not feed on the experimental host, and because the two hosts are so distantly related that they cannot be grafted on each other.

Host Ranges. Among the plant viruses we find some with very extensive host ranges, as in the case of the beet curly top virus that attacks 220 host species in 41 families of plants. Tobacco and cucumber mosaic viruses and aster yellows virus also attack many species often in widely separated families. At the other extreme are such viruses as that of wheat mosaic, which is restricted to the wheats, barley, and rye, or corn mosaic that attacks only corn and sorghum. Many viruses have been recorded from only a single host species, but we must always bear in mind the

possibility that virus diseases of two distinct plant species may be due to the same virus. Usually, but not always, there is some degree of botanical relationship among the plant species susceptible to a given virus.

Resistance to Heat. Plant viruses tolerate heating in varying degrees. On the one hand, tobacco mosaic virus, the most resistant of all, can stand 10 minutes of heating up to 198°F. before it is entirely destroyed. It has been known to tolerate the heating involved in the curing process. Spotted wilt virus of tomatoes and other plants is quite unstable. It is destroyed by heating for 10 minutes at 104°F. Indeed, if plants infected by this virus are placed in a hot greenhouse, they often lose the virus and recover. The other viruses that have been tested in this connection usually have heat tolerances between these extremes. Certain of the peach viruses can be removed from peach branches by heating, without injury to the peach tissues. This may have practical application in preventing virus spread by heat-treating propagation wood prior to use or shipment.

Resistance to Chemicals. In a similar way we find different degrees of tolerance of chemicals among the viruses, with tobacco mosaic virus topping the list in its resistance. In general, the viruses are quite sensitive to acids, alkalis, alcohol, and oxidizing agents. Frequently they are quite resistant to decomposition by enzymes.

Retention of Infectivity in Storage. The mechanically transmissible viruses, the only ones in which a study of resistance is satisfactory, retain their infective power in extracted juice for varying lengths of time ranging from a few hours to many months or even years with tobacco mosaic virus if the juice is kept from decomposition. At refrigerator temperatures, or when frozen solid, viruses remain viable for much longer periods than at room temperatures. Most viruses cannot stand desiccation, but tobacco mosaic virus in dried leaves or juice dried on filter paper, remains infective for many years.

Mutability. Many viruses exist in the form of various distinct strains, differentiated according to their different symptoms on one host plant or another. Indeed, some workers go so far as to feel that no two different field collections of a virus produce identical symptoms. In the case of tobacco mosaic virus, 50 or more strains have been distinguished. These agree for the most part in their physical and chemical properties and host ranges, but differ in their aggressiveness or in the symptoms produced on tobacco and other host plants. They include strains that produce no symptoms or very slight ones, strains that produce a moderate amount of mottling (mosaic) or distortion or both, strains that produce brilliant yellow or white mottling, strains that produce enations, and strains that result in moderate or extreme necrosis and death. Some remain

localized in the infected leaf, others move slowly or rapidly to the growing point of the plant and become systemic. This variation in viruses leads to great difficulties in virus recognition and classification.

Serologic Reactions. If a virus-containing juice is injected into the body of a rabbit, the rabbit's blood forms antibodies that will give precipitation reactions with the virus juice. The reactions are specific, i.e., the antibodies formed by inoculation of tobacco mosaic virus precipitate juices that contain various strains of tobacco mosaic virus but not other viruses, such as cucumber or potato mosaics. This affords a useful laboratory reagent for identifying unknown viruses and determining the relationships of viruses.

Acquired Immunity. Plants with systemic virus infection usually retain the virus as long as they live. Sometimes, after a wave of severe symptoms, the plants appear to recover and show no further obvious



FIG. 158. Recovery from a plant virus. (*Left*) This plant, after having experienced a severe case of tobacco ring spot, is recovering. New growth appears normal and will continue to be so in plants propagated by cuttings from the new growth. The recovered tissue still contains ring spot virus, but shows no further obvious symptoms in the new growth. (*Right*) This plant is an uninoculated control. (Courtesy, W. C. Price, Rockefeller Inst. for Med. Res.)

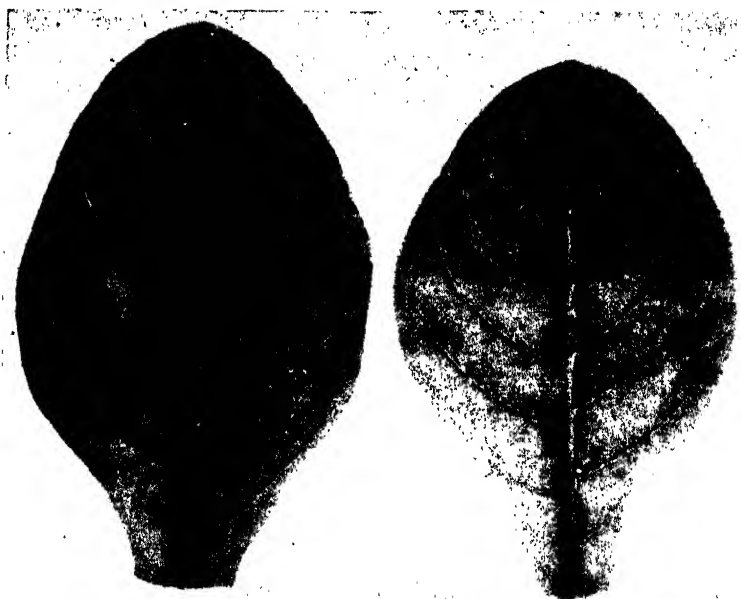


FIG. 159. Protective inoculation of a plant virus. The left half of the left leaf and the lower half of the right leaf of *Nicotiana sylvestris* were each rubbed with juice containing tobacco mosaic virus. Five days later the entire surface of both leaves was rubbed with juice containing the closely related tobacco aucuba mosaic virus. Aucuba mosaic lesions developed only on the halves that had not been protected by a previous inoculation of the non-necrotic virus. (Courtesy, L. O. Kunkel, Rockefeller Inst. for Med. Res.)

symptoms, although careful study may reveal abnormalities, such as pollen sterility (Fig. 158). These recovered plants contain the virus in virulent form. Their juice, if inoculated into healthy plants, produces the original disease. The recovered plants and their vegetative progeny are immune from further symptoms if again inoculated with the original virus, but are susceptible to other viruses. Because the acquired immunity reaction is specifically directed against the virus of the original infection, or its strains, the reaction is useful in determining virus relationships. Thus if a plant recovers from tobacco ring spot and proves to be immune from unknown virus A but susceptible to unknown virus B, we have evidence that A is a strain of the ring spot virus, while B is a distinct virus type. Similarly, a plant may be infected with a very mild strain of virus and thereafter prove immune from the severe strains of the same virus encountered in nature, analogous to smallpox vaccination with cowpox virus (Fig. 159). Whether or not mass immunization can be used to protect plants from virus diseases on a practical scale, remains to be seen, but there may be some possibility of this in vegetatively propagated plants like

potato where the immunizing virus would need be introduced only in the original breeding stock, or in mechanically transmissible viruses of plants like tomato and tobacco that are handled in transplanting, during which operation they could receive the immunizing dose from the operator's hands kept wet in virus juice. There are hazards, however, in such a control measure: the mild virus might mutate into a more destructive one; it might pass to some other crop where it could produce destructive disease; or it might associate with a second mild virus, the combination being quite harmful.

Nomenclature of Viruses

Prior to 1927, viruses were referred to by attaching the common name of the host to that of the principal symptom produced, as tobacco mosaic virus, peach yellows virus, or potato spindle-tuber virus. In that year J. Johnson proposed a system of numbering viruses, as tobacco virus 1, tomato virus 3, etc., a method that was further modified by Smith in England who substituted the Latin generic name for the common name of the usual host, as *Nicotiana* virus 1, *Prunus* virus 2, etc. This was followed by suggestions on using a Latin system of binomial nomenclature for viruses similar to that used for animals and plants, a proposal that was carried out extensively by Holmes who, in 1939, published a book assigning Latin binomials to large numbers of viruses. Here tobacco mosaic virus becomes *Marmor tabaci*, the genus *Marmor* including the mosaics, cotton leaf curl virus is *Ruga gossypii*, etc. The various virus genera are grouped into 10 families and larger groups, all plant viruses falling within the *Phytophagi*. Others have suggested somewhat different Latin terminology, but not extending it to a broad system, as Holmes has done. While Holmes' system is criticized in certain details by some of the present-day virus workers, there is likelihood that at least in modified form it will form the basis for future virus nomenclature as pathologists gradually familiarize themselves with the binomials.

Nature of Viruses

Ever since they were first recognized the nature of the viruses has been a challenge, and many theories have been advanced in attempts to answer the question: What is a virus? In the earlier days when potato degeneration, now known to be due to an accumulation of viruses, was a problem, some of the European workers advanced the hypothesis that the "running-out" of potato varieties was due to unbalanced nutrition. Others believed that it was caused by excessively long-continued vegetative propagation and the absence of stimulation from sexual reproduction. At the end of the

nineteenth century when the contagious nature of viruses was recognized, they were considered to be either filtrable bacteria (Iwanowsky) or a "contagious living fluid" (Beyerinck). During the succeeding years up till 1935 the majority of workers held that viruses are living organisms too small to be detected with the microscope, but in most other respects analogous to bacteria. This view was based on no direct proof, but on many suggestive characteristics of the viruses: their biological relationships with insects and incubation periods in insect bodies, their mutability, their specialized plant-host relationships, their reproduction, and their susceptibility to heat and chemicals, which is comparable to that of living pathogens. But not all virologists subscribed to this view. There were some who maintained that viruses may be chemicals analogous to enzymes, with the power of self-reproduction in living cells at the expense of cell materials. The ingenious theory has been advanced that viruses are "wandering genes," or factors of cell inheritance, perhaps segments of chromosomes, that have the power to pass from cell to cell and in each new cell propagate themselves and produce, by a genetic-regulatory mechanism, the symptoms of virus disease. Strange as this theory may seem, it has some support in the fact that viruses and genes are remarkably alike in their reaction to x-rays. Indeed, we have always conceived of genes as fixed components of cells, but were it possible for genes to pass from one cell to another their results might well be akin to those we see in virus diseases.

In 1935, Stanley prepared from tobacco mosaic virus juice a paracrystalline protein which he and others have regarded as the virus of tobacco mosaic in pure form. Crystalline proteins have been prepared from other virus juices but are not obtained in all cases. The question is not finally settled whether, in all cases, these materials are in reality the viruses or whether they may be closely associated by-products of virus activity. The most recent studies on virus proteins, although involving highly elaborate methods of accumulating data and interpretation, deal more with the chemical nature of these proteins than with their pathology, which in the end has always been the criterion of viruses.

A view that somewhat reconciles the biological and chemical theories is that the virus is part of a cell acting in the test tube as a chemical, not as a living organism, since it lacks the cell framework, but which, on introduction into a host plant or insect, takes its place as a functioning, misleading element of the living cell where, with the help of the other normal cell structures, it is able to reproduce itself. This approaches the "wandering gene" theory again.

The question of whether viruses are living or lifeless goes back in the

last analysis to a definition of life. Formulate a workable definition of life and it is at once possible to say whether or not viruses fit the definition. But it is not possible to formulate such a definition. As we pass down to the simplest living organisms each of the attributes of higher living creatures becomes subject to exceptions. For example, there are bacteria that do not respire by burning oxygen and liberating carbon dioxide, and one by one the other criteria of life fail as we approach the lowest limits of acknowledged living things. Again, on the chemical side we find the most complex chemicals displaying properties ordinarily associated with life, as the autocatalytic enzymes with the power of self-reproduction.

Perhaps the view of Andrewes and other British workers comes closest to the truth: that there is no sharp line that divides the living from the nonliving world. As we pass down the scale of life to the simplest living things—the iron and sulfur bacteria and the Rickettsia bodies that are too small to be seen with the ordinary microscope but are revealed by the ultramicroscope—we pass imperceptibly to the larger viruses, thence through to the smallest, as those of chicken cancer and bacteriophage, thence again imperceptibly into the most complex of chemicals, the self-reproducing enzymes with physiologic effects not unlike those of viruses, down through the growth-promoting substances and proteins, into the world of lifeless matter. The biologist, who regards the viruses as living, studies them in living hosts where they behave as organisms; the chemist, who considers them chemicals, studies them in the test tube where he sees only their chemical and physical properties. May not both be right, the viruses being only a connecting link that marks one step in a continuous and unbroken series from one kingdom to the other?

Specific Virus Diseases

TOBACCO (TOMATO) MOSAIC (*Marmor tabaci*)

Best known of all viruses is the common mosaic virus of tobacco which not only is a serious pathogen but also is the "guinea-pig" virus that has been of inestimable value in studies on the nature and properties of viruses in general.

History and Distribution. Tobacco mosaic virus occurs wherever susceptible plants will grow. In America it is everywhere a major factor in tobacco production except in the deep South where it may be masked by high temperatures. Moreover, it is one of the oldest recognized viruses. It was the first virus shown to be contagious (Mayer in Holland, 1886), the first found to pass through filters which hold back bacteria (Iwanowsky in Russia, 1892), the first to be extensively studied with regard to properties

and strains, and the first to be prepared in paracrystalline form (Stanley, 1935). The science of human and animal virology is much indebted to tobacco mosaic virus for providing basic information on this group of pathogens.

Importance. The losses in the nation's tobacco crop from mosaic range between 35 and 45 million pounds annually. If, as often occurs, plants become infected at the time of transplanting, the yield is reduced 30 to 35 per cent and because of poorer quality as well as decreased yield, the value of the crop per acre is reduced more than 55 per cent. If infection occurs a month later, the loss is nearly as great, and even late season infection (at topping time) significantly lowers the yield and quality of the crop. The losses in tomatoes are comparable.

Host Plants. Tobacco mosaic virus can attack 200 species of plants in 36 botanical families, with more than one-third of the recorded host plants in the *Solanaceae*. All tested species of tobacco are susceptible. Tobacco, tomato, pepper, and petunia are the most important economic hosts.

Symptoms. The most common symptoms of tobacco mosaic are leaf mottling of light and dark green patches (Fig. 160), leaf distortion,

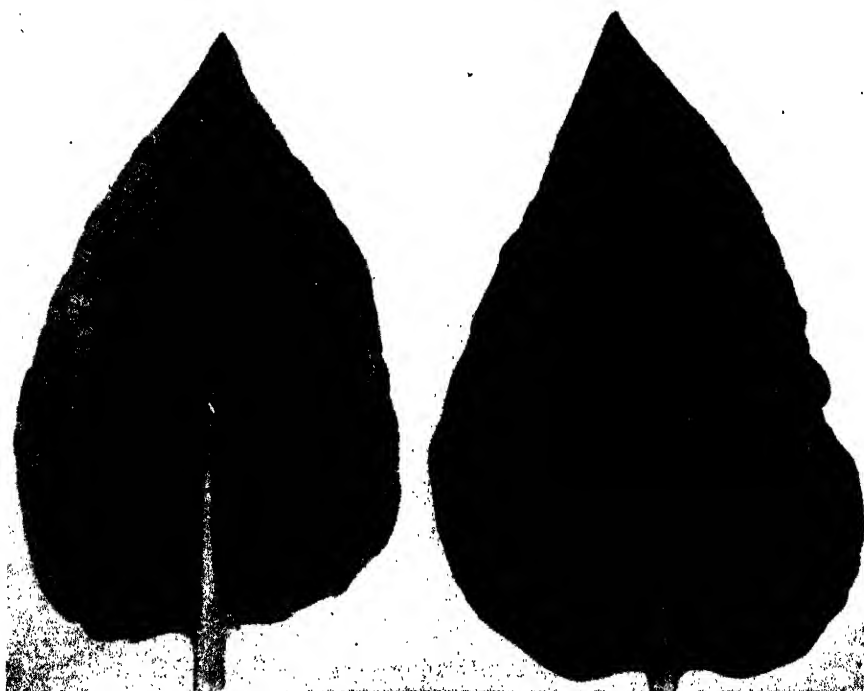


FIG. 160. Tobacco mosaic. (Left) Tobacco leaf affected with a moderately severe strain of mosaic. (Right) Normal leaf.

with blisterlike raised areas, and irregular or unnatural leaf shape, stunting of the entire plant, dwarfing, distortion, and variegation of the blossoms, production of mosaic suckers, and in some cases local or widespread necrosis. The symptoms vary with the virus strain and the host variety. Mild or symptomless strains have been isolated, as well as strains causing brilliant mottling or even death of the plant, although in general the disease is not a killing one. The symptoms are masked at high temperatures. If bean, *Nicotiana glutinosa*, or certain other plants are inoculated with the virus, primary infection takes the form of local necrotic spots from which the virus does not spread out into the growing point and other parts of the plant (Figs. 157, 159). In tomatoes the fruit may be mottled, and severe strains of the virus cause dead streaks on the stems or general necrosis of the plant.

Etiology. Tobacco mosaic virus is the most resistant and the most highly infectious of all viruses. It withstands heating almost to boiling, is quite highly resistant to alcohol and other germicides, and for many years retains infectivity in the dried state. The commonest original source of inoculum is smoking or chewing tobacco. Workmen using tobacco contaminate their hands with the virus and infect tobacco or tomato plants handled in operations of transplanting, pruning, disbudding, tying, topping, artificial pollinating, or harvesting. The virus is infective in high dilutions, up to 1:1,000,000, so that even a trace on the hands will serve to infect many plants. The virus enters plants through scratches, abrasions, or broken hairs, due to handling or rubbing the leaves. In ordinary tobacco, little evidence of disease is seen on the inoculated leaf, but staining with iodine shows that there is disruption of starch metabolism. The virus rapidly multiplies and passes to the growing point, largely by way of the phloem vessels. Here, in about 8 to 10 days after inoculation, are seen the first symptoms, a clearing-of-veins of the new leaves, and each succeeding leaf formed displays the characteristic mottling and distortion. The virus spreads from one plant to another in the field or greenhouse chiefly through the agency of man handling diseased, then healthy, plants, or by means of cultivators. The seed of diseased plants is not a source of infection. In spite of the high infectivity of the virus, plants normally do not pick it up directly from soil, but they may get it indirectly from the hands of laborers who have handled soil that is contaminated with virus-infested plant refuse.

Control: 1. **SANITATION.** In tobacco culture effective control is obtained by furnishing laborers with heat-sterilized or mosaic-resistant smoking or chewing tobacco and requiring that they use no unsterilized susceptible tobacco. Other important sanitary practices in growing to-

bacco and tomatoes include: washing the hands with soap or trisodium phosphate, before handling plants; handling healthy plants in the early part of the day and reserving work that involves handling diseased plants for the last thing in the day's work; weeding out and destroying infected plants as they appear; burning of mosaic-infested crop refuse; and avoiding unnecessary or excessive handling of plants. In the greenhouse there are many fine points of sanitation. For example, the doorknobs, faucets, pots, flats, and hose-ends may become contaminated by handling with virus-smeared hands, and thereafter serve as unsuspected sources of infection. The vibrating pollinator is particularly dangerous. A combination of routine sterilization of equipment and soil, devices for avoiding contamination, such as foot- or elbow-operated faucets and doors, and above all, vigilance in detecting and eliminating dangerous practices will protect the greenhouse from mosaic.

2. BREEDING FOR RESISTANCE. No mosaic-resistant commercial tomatoes have yet been produced, but resistant breeding stocks are available. On the other hand, recent work has incorporated mosaic resistance into commercial tobacco and peppers. In tobacco, the principal source of resistance has been *Nicotiana glutinosa*, a wild species in which the virus forms a small local necrotic spot at the point of infection and does not usually spread beyond this point. Crossing this with commercial Burley and dark fire-cured tobaccos, and then backcrossing with the commercial types has produced mosaic-resistant commercial tobaccos that are now being grown on an increasing acreage, including Ky. 34, 52, 150, 151, and 160.

BEAN MOSAIC (*Marmor phaseoli*)

There are several virus diseases of garden and field beans of which the most widespread is the common bean mosaic. It is of worldwide distribution, occurs throughout the United States, and ranks with anthracnose and bacterial blight as a leading bean disease. Bean mosaic exhibits many of the characteristics of a group of related mosaic diseases attacking cowpeas, soybeans, mungbeans, and other legumes.

Symptoms. The virus is systemic, distorting, yellowing, and stunting the plant but rarely killing it. Mottling of light and dark areas on puckered, downward-cupped leaves is typical, but the mosaic pattern is not so clearly defined as in tobacco and other mosaics, and can easily be confused with drought or insect injury. (Fig. 161.) The dwarfed plants produce lowered yields, or if attacked early the yield may be almost nil, but in the beans that are formed no certain symptom of the disease is recognizable although the beans are often undersized or shriveled. The

disease is somewhat unusual in that the symptoms are not masked but exaggerated at high temperatures.

Etiology. Bean mosaic is caused by the virus *Marmor phaseoli*, and the varieties of common bean are the only hosts. The virus is average in its resistance to heat, being destroyed at about 134°F. in 10 minutes. It may with difficulty be transmitted to healthy plants by rubbing them with juice from diseased plants, and carborundum powder is added to the juice to

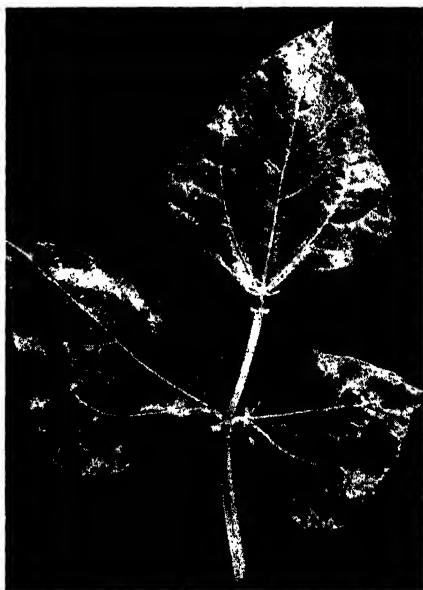


FIG. 161. Bean mosaic. The symptoms are dwarfing, distortion, and light green mottling of the leaves, resulting in poor yields.

favor artificial inoculation by causing small leaf abrasions. In nature, the virus is not spread by handling plants, but is transmitted by a number of species of aphids. This is one of the cases, unusual except among the legume mosaics, of a seed-borne virus. Thirty to 50 per cent of the seed from diseased plants produce mosaic diseased seedlings, and from these the aphids soon carry the virus to entire plantings. Some varieties of beans, notably nonresistant strains of Refugee, have been so generally infested that commercial lots of seed regularly have contained the virus.

Control. Use of mosaic-resistant bean varieties is the most effective way of preventing losses from this disease. Of the older standard varieties, only Robust was resistant, but breeding, particularly in Idaho, has given us a number of newer resistant varieties of different types, including the Great Northern types U. I. 1, 15, 59, 81, and 123, Red Mexican U. I. 3 and

34, Wisconsin Refugee, Idaho Refugee, U. S. No. 5 Refugee, Sensation Refugee 1066 and 1071, Meda1 Refugee, Michelite, Florida Belle, Florida White Wax, and Logan. Removal of diseased plants as soon as they can be recognized is helpful in cleaning mosaic out of valuable seed stocks but is not practical on a large scale. The virus cannot be removed from seed by seed treatment. It is very hazardous to plant a fall crop of susceptible beans after a mosaic-affected spring crop; the fall crop is likely to be a total loss from mosaic.

WHEAT MOSAIC (*Marmor tritici*)

Wheat mosaics have been reported in several Great Plains, central, and eastern states. Once established the trouble often becomes very destructive.

There are several viruses affecting wheat, producing some combination of the following symptoms, usually most evident in the spring: mottling or variegation, the leaves showing a pattern of light and dark green or green



FIG. 162. Yellow mosaic in Harvest Queen wheat. The four leaves at the left are from plants in the tiller-formation stage, the two at the right are from plants in the jointing stage. The lightest areas in these leaves were yellow or almost white. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

and yellow patches or stripes (Fig. 162); rosette or stunting with excessive production of small tillers and lack of normal jointing; dead leaves at the base; rolling of the leaves as in wild garlic; and occasionally killing of the plants. Attacked plants sometimes recover spontaneously. The trouble often has a tendency to appear in well-defined spots in the field, although in light attacks only single plants here and there may show the symptoms.

The wheat viruses of the eastern states appear to differ from those of the Great Plains in that the eastern viruses are perpetuated from one crop to the next in the soil, where they can survive up to six years, while there is no positive evidence of this in the western viruses. Spread in the field probably is through the agency of insects, although data on this point are scanty. Volunteer grain or grasses may possibly serve for the overwintering of the viruses.

Comparatively few data are available on which to base recommendations for the control of the wheat mosaics west of the Mississippi River. The eastern mosaics are readily controlled by resistant varieties of wheat, many of which are available. In the West the disease is erratic in appearance, which hampers varietal resistance tests. In lack of specific control measures it would be advisable to plant infested land with some cultivated crop other than wheat or rye for at least one year. Where the disease is known to occur, seed might be selected from fields known to be free from the disease. Observations on the varieties that fail to be attacked in the localities of infestation will be a guide for future plantings. Mosaic-resistant soft red wheats under conditions east of the Mississippi include Prairie, Michigan Amber, Rudy, certain lots of Fulcaster and Fultz, Gladden, Forward, two selections of Jones Fife, Mediterranean, Poole, Red Rock, Red May, Red Wave, Trumbull, Shepherd, Prairie, Nabob, Wabash, Thorne, some selections of Harvest Queen, and Fulhio. The more resistant hard red winter wheats are Cooperatorka and Eagle Chief, with Oro and numerous other winter wheats partly resistant. Such commonly used varieties as Kawvale, Turkey, Cheyenne, Tenmarq, standard Harvest Queen, and Clarkan are not resistant.

SUGAR CANE MOSAIC (*Marmor sacchari*)

This is an outstanding case of a disease of such destructiveness that it all but wiped out American cane sugar production, yet which, by breeding for disease resistance, has been brought under control, with restoration of the industry. Mosaic reduces the yield of sugar in affected plants by amounts ranging up to 40 or 60 per cent. Since this is a crop that is propagated vegetatively, by stalk pieces, virus-infected canes produce infected progeny, and this, together with spread in the field by aphids, can

result in steady increase of mosaic until plantations become unprofitable. The virus is not easily transmitted by mechanical methods.

Sugar cane mosaic causes a chlorotic streaking of the leaves and stunting of the plants. There are several virus strains concerned, producing somewhat different types of mosaic pattern and effects on yield. The common type also affects corn, sorghums and a number of grasses. Control is based on use of resistant varieties, roguing mosaic out of seed plots, and isolating cane from corn and grasses that harbor the aphid vectors. A search for resistant sugar cane throughout the world brought to light some promising strains, and these have been improved and propagated until they now occupy the greater part of the commercial sugar cane acreage, and the great destructiveness of the disease is a thing of the past. Further details on this disease are given by Tims *et al.*, cited in the references at the end of the chapter.

CUCURBIT MOSAIC (*Marmor cucumeris*)

This is a widespread typical mosaic disease of cucumber, squash, celery, spinach, tobacco, petunia, pepper, and many other species (Fig. 163). The virus causes the important spinach blight disease, and takes part in a destructive complex of viruses in lilies and other ornamentals. It overwinters on a number of weed species. The virus is easily transmitted mechanically and is carried also by aphids and cucumber beetles. It is controlled by cutting diseased plants at the base, to avoid spreading the disease by pulling them out, eradication of weed hosts such as milkweed, pokeweed, wild cucumber, and ground cherry, and by fumigation or



FIG. 163. Cucurbit mosaic. (A) Symptoms of distortion, chlorosis, and stunting in muskmelon. (B) Mosaic on the cucumber fruit, also known as "white pickle." (Courtesy, C. T. Gregory, Ind. Agr. Extension Service.)

spraying to kill the aphid vectors. An active program of testing and breeding to develop mosaic-resistant varieties of the various crops affected is being carried out. This has given us the resistant Ohio 31 and Maine No. 2 cucumbers, the Geneva Delicata and Shamrock squashes, and Old Dominion, Virginia Savoy, and resistant Bloomsdale spinach. Sources of mosaic resistance in other crops, such as muskmelon, are known and their use in breeding probably will furnish more mosaic-resistant varieties in the future.

ASTER YELLOWS (*Chlorogenus callistephi*)

While tobacco mosaic virus has been a classic object for studies on the nature and properties of the mechanically transmissible, mosaic-type viruses, the aster yellows virus has served similarly to provide an understanding of yellows-type viruses. Usually these differ from the mosaic viruses in their inability to be transmitted from plant to plant by juice inoculations, but only by specific insects in which the virus has an incubation period, or by grafting or use of dodder bridges.

Host Plants. First known as a pathogen of the China aster, in which it has been very damaging, the aster yellows virus has since been found to cause important diseases of 170 or more species in 38 families of plants, including lettuce, celery, salsify, parsley, mustard, dill, carrot, buckwheat, parsnip, New Zealand spinach, and many ornamentals and weeds. In some of these, such as carrots and lettuce, aster yellows is a major disease. In potatoes the virus causes the purple-top wilt disease. Very common weed hosts are the daisy fleabanes, species of *Erigeron*, and wild lettuce.

Symptoms. In most hosts, the disease first shows as a clearing of veins of new leaves formed after inoculation. This is followed usually by an even yellowing of newly formed tissues and stimulation of normally dormant buds to produce branches, which leads to dense witches' brooms of many weak branches. The plants have an abnormally upright habit and stiff structure, and are frequently sterile (Fig. 164).

Etiology and Epiphytology. Aster yellows is caused by the virus *Chlorogenus callistephi* var. *vulgaris* and, in California, a closely related virus, *C. callistephi* var. *californicus*. The properties of the virus are largely known because of the classic studies of Kunkel, cited in the references at the end of this chapter. The virus is transmitted in nature by the leaf hopper *Cicadula sexnotata* and less frequently by other leaf hoppers. In its vector the virus has an incubation period of from 17 to 26 days. By delicate microinoculation methods at freezing temperatures Black, an associate of Kunkel, was able to transfer the virus from one leaf hopper to another and prove that the virus multiplies at least a hundredfold in the insect's body, but many attempts to transfer the virus by juice from one plant to another

have failed. Kunkel has been able to cure yellows-affected plants and to free leaf hoppers of the virus by heat treatments which did not injure the plants or insects.

In nature, infected leaf hoppers which have picked up the virus from wild hosts or cultivated plants, are the means of spread of the disease.

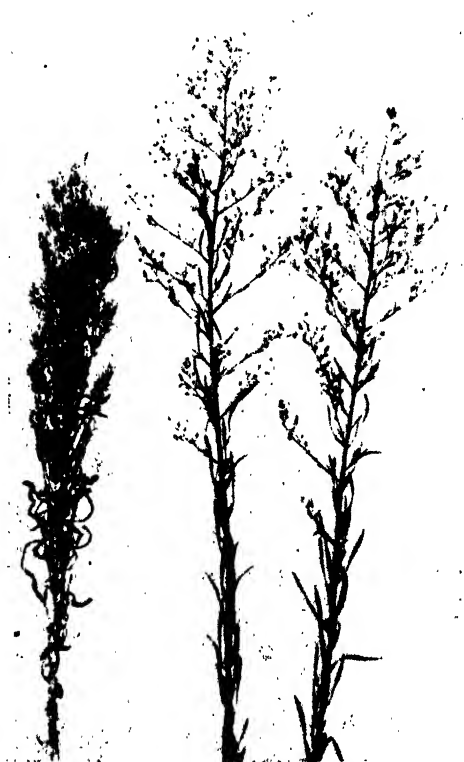


FIG. 164. Aster yellows in the weed, daisy fleabane (*Erigeron*). The dense witches' broom of weak and sterile branches (*left*) replacing the normal flowering panicle (*center and right*), is typical of the effects of aster yellows virus in various hosts.

These insects do not rise high above the ground, and Kunkel showed that a fence of fine mesh screen six feet high around plantings will protect the plants within the fence against leaf hoppers.

Control. Since the prevalence of the disease is determined by the population of infected leaf hoppers, control measures are directed against them by use of screen fences or cages; where the value of the crop justifies this, and eradication of weeds on which the leaf hopper feeds and over-



FIG. 165. Curly top of sugar beets. Natural infection, showing the inward curling of the leaves toward the midrib. (Courtesy, H. H. P. Severin, Cal. Agr. Exp. Sta.)

winters. In commercial aster production screen cages are used extensively. Spraying for leaf hopper control has not been very helpful. Prompt roguing of the first affected plants is useful in reducing later infestation. Control by the use of resistant varieties has not made much progress.

CURLY TOP OF SUGAR BEET (*Ruga verrucosans*)

This represents a third class of virus diseases, the leaf curl group. Curly top has caused a very serious drain on the sugar beet industry. When the disease became established west of the Rockies it was so destructive that many thousands of acres of beets were abandoned, sugar factories were closed, fine farming areas gave up trying to grow sugar beets, losing an important cash crop and one greatly needed for the best diversified farming. Recovery of that industry through the development of high-yielding, curly top-resistant beet varieties is another of the triumphs of plant breeding (see Frontispiece).

Host Plants and Symptoms. The appearance of a beet plant with curly top is shown in Fig. 165. In early stages there is vein clearing followed by rolling and curling of the leaves and many wartlike swellings which roughen the undersides of the leaves. Frequently the leaves become yellowish. Root development is checked, with degeneration of the phloem, and the plants die prematurely until, in severe attacks, fields are almost barren, with yields reduced 80 to 100 per cent.

Tomato is another important host of the curly top virus. Here the disease is called western yellow blight or yellows. The symptoms are a retarding of growth, dropping of blossoms and buds, rolling, yellowing, and thickening of leaves, and root decay, usually followed by death but sometimes by recovery. The curly top virus causes diseases of importance in numerous other crops, among them beans, table beets, spinach, squash, peppers, and alfalfa. In all these the symptoms are similar to those described above. Of the many weed hosts the Russian thistle and wild mustards particularly are important.

Etiology and Epiphytology. Curly top is caused by the virus *Ruga verrucosans* which is normally transmitted by the beet leafhopper, *Eutettix tenellus*. The range of this insect in the western states limits the disease to the same area. Mechanical transmission of the virus is possible, but difficult. In the insect vector the virus has a short incubation period, 4 to 12 hours. In the plant the virus is confined to the phloem, which leads us to the next type of virus disease to be studied, phloem necrosis, and suggests why the beet leaf hopper, a phloem feeder, is the vector. The virus is quite resistant to disinfestants such as alcohol and mercuric chloride, and survives heating up to 167°F. and dry storage for from 6 to 10 months. This indicates that the difficulty in mechanical transmission is not due to instability of the virus.

The virus overwinters in surviving leaf hoppers that have become infected through feeding on infected beets or weed hosts in summer and fall. Beet plants are most susceptible in the seedling stage, and weather that favors overwintering of the insects or that delays planting or hastens movement of infected insects into the field favors epiphytotic development of curly top. This is a reason why the disease is very destructive some years and mild during others.

Control. Measures to fight or avoid the leaf hopper have been of some help, but the real solution of the curly top problem has been the development of resistant beet varieties. Chemical methods of leaf hopper control have not been very successful, but a thorough study of the insects' habits has made it possible to forecast curly top outbreaks in time to escape the disease by substitution crops during the worst curly top years. There has been some success in leaf hopper control by wholesale eradication of the Russian thistle. Early planting to get the crop through the most susceptible period before the leaf hoppers move into beet fields also has been very helpful. The combination of these practices and the use of resistant beet varieties has been the most successful control program.

The first curly top-resistant sugar beet, U. S. No. 1, had only moderate resistance, and suffered under severe disease outbreaks, but greatly out-

yielded the old type susceptible varieties. Selection of the more resistant individuals of U. S. No. 1 produced strains with increasingly high resistance. The yields of these, in comparison with the susceptible old type beet during a year of moderate curly top (1940) and one of severe disease (1941) are given in Table 6.

Table 6

YIELDS OF SUSCEPTIBLE AND RESISTANT SUGAR BEETS
DURING CURLY TOP OUTBREAKS AT TWIN FALLS, IDAHO

Variety	Yield in tons per acre	
	1940	1941
Old Type.....	5.16	0.00
U. S. 1.....	..	6.31
U. S. 33.....	19.26	8.40
U. S. 12.....	..	11.25
U. S. 22.....	27.09	14.32
Improved U. S. 22.....	29.39	16.61

The resistant beets also are so satisfactory in sugar yield and other desirable characters that they restored the sugar beet industry of the West, where resistant varieties are now grown almost to the exclusion of the old type beets.

In Utah and other states where curly top sometimes is the limiting factor in tomato production, the losses are reduced by certain cultural procedures, by planting at close intervals or by setting two plants to each hill, so that if one is lost from curly top the other may still produce. The extra cost of planting is offset by increased yields during curly top years. Sometimes shading the plants or growing them under muslin cages for protection from leaf hoppers is practiced. Breeding for curly top resistance is in progress with South American tomato species and novelty tomato varieties serving as sources of resistance. Similar work is being done with beans, and the curly top-resistant strains U. I. No. 3, 34, and 15 have been developed. The last is a Great Northern type of bean with resistance to both curly top and mosaic.

PHLOEM NECROSIS OF ELM (*Morsus ulmi*)

Viruses in plants are active particularly in the phloem which they often use as a path for spread through the plants. At times this activity results in a killing of the phloem tissues. We have already seen evidence of this in the degeneration of the phloem of beets attacked by curly top. There are several virus diseases in which necrosis of the phloem is an outstanding

symptom, and the phloem necrosis disease of the American elm illustrates this type of virus disease.

Importance; Range; Hosts. This disease probably has been present in America since 1880, and since 1918 it has spread rapidly, destroying many thousands of valuable elms in the Corn Belt. Some communities have lost 80 per cent of their trees, and within a single year 10,000 trees were killed in Columbus, Ohio, and 20,000 in Dayton, Ohio. From its initial occurrence in Illinois and Ohio, phloem necrosis has spread westward to Oklahoma, Kansas, and Nebraska, north into Iowa, south into Arkansas, Mississippi and Tennessee, and east as far as West Virginia. No one knows how many more thousands or millions of trees may be destroyed before this almost uncontrollable disease has run its course. Only the American elm is attacked; European and Asiatic elms, including Chinese elm, are resistant.

Symptoms. Symptoms develop in inoculated trees in from six months to three years. A slight wilting is noticed first, soon followed by yellowing and falling of leaves, which increases until the tree may be bare, followed by its death. Other causes may produce similar defoliation, and the most reliable diagnostic character is detected by cutting a chip from the trunk base. Here the inner bark (phloem) is found to be yellowish to brownish, instead of normal white. The scraped-off phloem of diseased trees, when enclosed and warmed, has a characteristic odor of wintergreen. The destruction of the phloem causes the bark to loosen and fall away when infected trees die, which regularly occurs a few weeks or months after the symptom of wilting first appears.

The root system suffers particularly. The fibrous roots die, necrosis progresses up into the larger roots and finally, after death of the roots, the phloem of the lower part of the stem is killed. It is this root failure, due to the inability of food to pass to roots through the injured phloem, that leads to wilting, defoliation, and death.

Etiology and Epiphytology. The disease is caused by a virus, as shown by grafting experiments. Natural spread is probably by insects, although the vector is not known. Some spread occurs from tree to adjacent tree by natural root grafting. In this way the disease may spread down a row of street trees.

In its invasion of new areas the disease first affects small, widely separated groups of trees. These become more numerous and larger until the disease occurs uniformly over the area. There are periodic outbreaks in which the disease kills many trees in a season, followed by a subsidence, and then, later, new destructive outbreaks. This rise and fall of the disease may be related to fluctuations in the population of an insect vector.

Control. Little can be suggested for controlling phloem necrosis. Infected trees, as soon as recognized, should be removed to check the spread. Some communities in the path of the spread have laid up large supplies of DDT with the purpose of thoroughly spraying all town elms on first appearance of the disease, in the hope that the insect vectors may be controlled. In other communities street elms are being interplanted with young elms of resistant species, sycamores, or other resistant trees, so that if the large trees are destroyed, a substitute planting will be rapidly developed.

At the Forest Pathology Laboratory in Columbus, Ohio, many thousands of elms have been tested and some have withstood repeated inoculation with the virus. These are hybrids of American elm with Chinese and other elms, which may be resistant and of a desirable type for street planting. Also, some American elm trees in the path of the disease have survived where nearby elms have been destroyed. From these sources there is hope that resistant elms of suitable form and adaptation may eventually be selected and propagated to replace the trees that have fallen victim to phloem necrosis.

CITRUS PSOROSIS OR SCALY BARK (*Rimocortius psorosis*)

In many orange, lemon, and grapefruit orchards in California and Florida heavy losses have resulted from the attack of psorosis virus which reduces tree yields about one-third and eventually kills the trees. The disease has some unusual features of symptoms, spread, and control, and serves to represent a fourth type of virus disease, one in which bark symptoms predominate.

Symptoms. On leaves of newly infected trees there is first a transient clearing of veins, yellow flecking, or yellow oak leaf pattern. In one type of psorosis older leaves and fruits show chlorotic or necrotic rings. Later the bark of trunks and branches becomes flaky or scaly, at first locally and superficially, but then the lesions extend deeper, encircling the trunk or branch and spreading in a systemic fashion, at which time the tree rapidly deteriorates (Fig. 166). There may be a gummy flow in the lesions, and these often are invaded by fungi which contribute to the damage. In the inward extension of the lesions, the water-conducting vessels are plugged with gum, resulting in water shortage which is the main factor in tree deterioration.

Etiology and Epiphytology. The psorosis virus, of which there are two strains, is transmitted chiefly by man in the propagation of trees by budding. It can be experimentally transferred by bark grafting—a useful technique to determine whether or not budwood trees are infected. In

nature it sometimes spreads from one tree to the next by natural grafts of the roots, but beyond this no insect vector or other efficient means of natural spread is known.

Control. Psorosis is controlled by eradication or treatment of diseased trees and avoidance of disease spread in propagation. Trees with advanced



FIG. 166. Bark lesions and wood stain of citrus psorosis. (Courtesy, Dr. H. S. Fawcett, Citrus Exp. Sta., Riverside, Cal.)

infection must be removed. In early stages, before the disease becomes systemic, trees may be cured by removing infected branches, by scraping out the lesions, or by treating them with Dinitro. The California Department of Agriculture is pioneering in a service which may have future application in the production of other virus-free tree crops. Trees to provide budwood are regularly inspected and tested for freedom from psorosis by bark patch grafting. These proved disease-free trees are

registered as certified sources of virus-free buds. Orchardists pay a premium for their progeny, with which they receive written statements of budwood origin. Several thousand such budwood trees now bear registration numbers so that a good supply of disease-free budwood is available to growers.

VIRUS DISEASES OF POTATOES

In the United States there are 14 typical potato viruses, 10 more which usually are associated with other hosts, and a number of other diseases of potato of suspected virus nature. Some of these viruses are present in the form of numerous distinct strains, and they include viruses of the several types already studied. The most important of the potato viruses and the diseases they cause may be characterized as follows:

Latent Mosaic Virus (*Marmor dubium* = X-virus of European workers = "healthy potato virus"). Although less common in other parts of the world, the latent mosaic virus occurs in practically every plant of some of the commercial potato varieties grown in America. In most potato varieties it produces no noticeable symptoms but when the yields are measured it is found that the virus has insidiously lowered the production by 9 to 22 per cent. When plants with latent mosaic become infected by a second virus, in itself comparatively harmless, the combination of the two viruses may produce serious disease. The latent mosaic virus is easily detected in potatoes by rubbing a little juice of an infected potato plant on the leaves of a pepper seedling, where it produces an extensive necrotic disease. The virus is very easily transmitted by mechanical methods and is spread by the seed-cutting knife. It ranks second to tobacco mosaic in infectivity, and the virus juice may be diluted 1:10,000 and still produce infection. Its host range includes pepper, Jimson weed, henbane, tomato, tobacco, bittersweet, nightshade, amaranth, chrysanthemum, and speedwell. The latent mosaic virus combines with tobacco mosaic virus to cause *double-virus streak*, a necrotic disease of tomatoes, and with the vein-banding virus of potato to produce the important *rugose mosaic* of this crop.

Vein-banding Virus (*Marmor cucumeris* var. *upsilon* = Y-virus of European workers). This causes no symptoms in some potato varieties, and in other cases either a pale mottling or leaf-drop and a stem-streak. By itself it is not a major problem but in combination with the latent mosaic virus it produces *rugose mosaic*, one of the most destructive of the potato virus diseases. The vein-banding virus occurs widely in the United States and Europe. Its host range includes a number of the other members of the potato family and the cowpea, on which it produces characteristic red local lesions. It is easily transmitted by mechanical means, and also is

carried by the aphid, *Myzus persicae*. The combination disease, rugose mosaic is a striking one (Fig. 167). The leaves are dwarfed, much wrinkled (rugose), and somewhat mottled, with a downward rolling of the leaf margins. There may be more or less necrosis of the stem tissues and of the veins. Affected plants are stunted and die prematurely so that tuber production is greatly reduced or even lacking.

Mild Mosaic Virus (*Marmor solani* = A-virus of European workers). The mild mosaic virus, which affects only potato and a few related species, usually causes a very mild yellowish mottling of potato leaves with some crinkling or wrinkling of the leaf surfaces. Diseased plants are slightly to severely stunted, and die prematurely with yields reduced about one-third.



FIG. 167. Rugose mosaic of potato. (Courtesy, J. M. Raeder, Idaho Agr. Exp. Sta.)

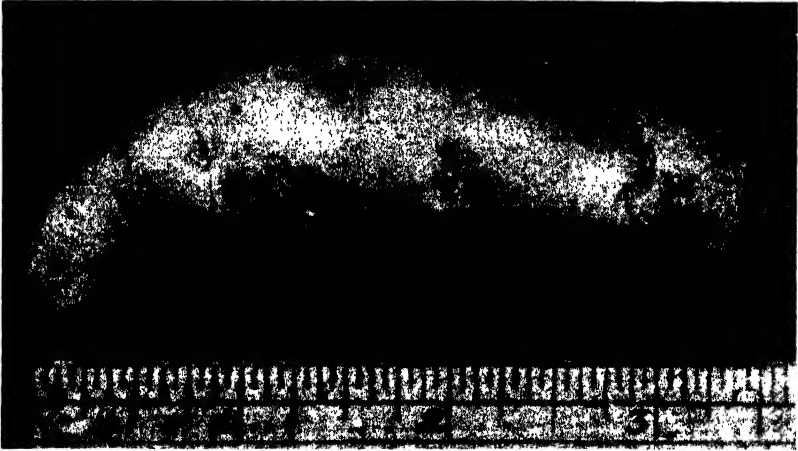


FIG. 168. Spindle tuber of Irish potato, one of the few virus diseases that can be recognized in the tuber. Off-shaped tubers such as this should not be planted. (Courtesy, C. D. Burke, Pa. Agr. Extension Serv.)

The disease is widespread in the United States and ranks among the major virus diseases of potato both because of its prevalence and because it may cause substantial losses that are largely unrecognized because of the mild foliage symptoms. The virus is mechanically transmissible but less easily than the viruses of latent mosaic and vein-banding. In nature it is transmitted by two species of aphids.

Spindle Tuber Virus (*Acrogenus solani*). This virus which affects only the potato, is widespread in the United States and Canada. Affected plants are stiff, with upright, and small, dark leaves. The tubers are abnormally long, spindling, and tend to be pointed especially at the stem end (Fig. 168). In the Bliss Triumph variety, the spindling shape is not so apparent. The tubers are small, often of a rounded conical form, bleached at one end, and with an excessive number of large, shallow eyes rather evenly distributed over the surface. Yields are reduced 60 or 65 per cent. Mechanical transmission of the virus occurs easily by leaf juice and by the seed-cutting knife, which is one of the chief means of spread of this virus. In the field it is transmitted also by aphids, grasshoppers, flea beetles, potato beetle larvae, and leaf beetles.

Leaf Roll Virus (*Corium solani*). The leaf roll virus affects only potato and a few closely related plants. It is found wherever potatoes are grown and differs from all the viruses previously discussed in that in nature it cannot be transmitted mechanically but only through its insect vectors. These vectors are three species of aphids, principally the peach aphid, *Myzus persicae*. In the insect, the virus has an incubation period of from 24 to 48 hours. The leaf roll virus causes one of the most destructive

of the potato diseases. Affected plants are pale and dwarfed, their leaves thick and leathery and rolled upward (Fig. 169). In some varieties the leaflets are reddened or purplish. Tubers are small and few and as the infestation may reach 25 per cent or more in the field the losses are heavy. In comparatively recent infections of some varieties of potatoes, the cut tubers often show a network of brown strands, especially near the surface and stem end of the tuber. This is *net necrosis*, one of the few easily recognized tuber symptoms of potato virus diseases in stocks that have not had the disease for over a year. It must be distinguished from similar conditions due to freezing, leaf hopper attack, and stem-end browning of unknown cause. The leaf rolling condition, too, might be confused with similar symptoms caused by genetic faults or unfavorable environment.



FIG. 169. Potato leaf roll. (Courtesy, J. M. Raeder, Idaho Agr. Exp. Sta.)

Other Potato Viruses. There are a large number of other viruses affecting potato in local areas or in less severe fashion than the foregoing. Among them may be mentioned: witches' broom virus that causes such excessive branching that the plant becomes a great mass of weak shoots; yellow dwarf virus, which is very destructive locally in the Eastern states, completely destroying the plants; purple-top wilt caused by the aster yellows virus; and calico and aucuba mosaic viruses that cause brilliant yellow and white mottling of the potato foliage.

Control of Potato Virus Diseases. Except for a few minor differences all the potato viruses are susceptible to similar control measures, viz.:

1. **USE OF VIRUS-FREE SEED TUBERS.** All potato viruses are carried in the tubers, usually without tuber symptoms that would enable the grower to detect virus-diseased tubers. The best assurance in protecting against these diseases is to use certified potatoes for planting, because the fields producing certified potatoes have been inspected for virus diseases, and any considerable amount of these diseases in the field disqualify it for certification.

In production of certified potatoes, the foundation stock is obtained by *tuber-indexing* or *hill-indexing*. An eye from each potato or a tuber from each hill is planted early or in the greenhouse. If the resulting plant shows virus symptoms the potatoes from that hill are discarded. After planting, the potato seed plot is inspected frequently and any hills showing virus disease symptoms are destroyed. This roguing of diseased hills is aided by the *tuber-unit method* of planting, in which the seed pieces from each potato are planted consecutively in the row, the end of the unit of four hills being marked by a missed hill or stake. Whenever a virus-diseased hill is found, the unit of four hills is destroyed. The procedure followed in Nebraska in the production of certified seed is representative: The first year's plants are raised from indexed tubers; this crop is multiplied the second year as a foundation seed plot, rogued as necessary; the third year the crop is planted by the certified seed grower in a tuber-unit seed plot; the fourth year's crop is then certified.

Certified potatoes are available in several grades. All grades are equally free from virus diseases. They differ principally as to the size, shape, and appearance of the tubers, and their degree of infestation with scab and *Rhizoctonia*, diseases that can be recognized on the tubers and controlled by seed treatment.

For southern growers it is usually impractical to produce their own seed tubers. In cooler climates this can be done, the home-grown tubers having all the advantages of certified seed tubers provided that a relatively

virus-free seed stock was used originally, and that the seed block is isolated, and thoroughly rogued several times during the season.

2. SEED INSPECTION. Occasionally virus diseases can be detected in the tubers: in spindle tuber and the net necrosis phase of leaf roll; potato yellow dwarf can be recognized in severely diseased tubers by rusty spots in the flesh. Such tubers should be rejected. Because spindle tuber is spread so easily by the cutting knife it is a good practice to use in rotation a series of knives standing in alcohol or formaldehyde solution.

3. FIELD PRACTICES. There is little that can be done in the commercial field once virus diseases have appeared. Elimination of potato dump piles (p. 217) reduces the populations of insect vectors of virus diseases. Sucking-insect control with contact insecticides may be tried to prevent field spread, but this has doubtful value except in seed plots. Roguing in commercial fields is undesirable as this merely reduces yields without appreciable advantage. Rotation is of no importance in this connection, because potato viruses do not live over in soil. Weed control may have some value in reducing sources of infection, but hardly enough to justify more than the ordinary cultivation practices, except in the case of seed plots where weeds of the potato family should be eliminated from adjacent fence rows. Early harvesting is an aid in reducing the percentage of virus in seed potatoes. This is aided by killing the vines with an herbicide. These chemicals are useful also for suppressing weed hosts of the virus vectors.

4. RESISTANT VARIETIES. A number of potato varieties have resistance to one or more of the potato virus diseases. Among these are the following. Resistant to mild mosaic: Chippewa, Erlaine, Irish Cobbler, Katahdin, Sebago, Sequoia, Warba, Houma; to mosaics in general: Katahdin, Chippewa, Sebago; to the vein-banding virus: Katahdin, Chippewa, Green Mountain, Russet Burbank; to purple-top wilt: Cobbler, Mesaba, Erlaine No. 2, Sebago, Pontiac; to yellow dwarf: Russet Burbank, Warba, Sebago, Houma; and to leaf roll: Katahdin, Sequoia, and Potomac. A potato strain, S41956, is resistant to latent mosaic, and this and other breeding stocks are being used in extensive potato breeding programs in America and England, in an endeavor to combine resistance to the several major potato diseases in single, acceptable varieties.

VIRUS DISEASES OF STONE FRUITS

Peach and related fruits have suffered long from virus diseases. The best known of these, peach yellows, has been an important factor in American peach production for 150 years. By 1936 some half dozen peach virus diseases had been recognized, but during the succeeding decade the number of stone fruit virus diseases multiplied to such an extent that by

1946 as many as 50 virus diseases of peach and the related stone fruits may be found described, a situation so complex that only a few stone fruit virus specialists are able to give an authoritative diagnosis of these troubles.

There are common characteristics which permit us to consider these stone fruit viruses as a group. Generally speaking, they devitalize the trees, lowering the quantity and quality of the fruit without killing the trees; they spread from one area to another in nursery stock and from one tree in the orchard to another, so far as is known, by sucking insects; they are controllable only by nursery stock control and prompt eradication of diseased trees, both of which procedures are aided by state and federal provisions for inspection, eradication, and quarantine. A few of the leading stone fruit viruses will be described and control measures summarized to illustrate the principles of dealing with this group of diseases.

Peach yellows (*Chlorogenus persicae* var. *vulgaris*), known in the United States since 1791, affects peach, nectarine, almond, apricot, and plum, the last being important as breeding sources of insect vectors and as symptomless carriers of the yellows virus. The disease occurs from Canada to the Carolinas and westward through the Corn Belt to the Mississippi River. Affected trees have abnormally upright wiry shoots in witches' brooms with upright side branches (Fig. 155). These develop prematurely in the spring with small, yellow, often red-spotted leaves that fold upward and curl back like a claw. The fruit ripens prematurely, and is abnormally large, watery, of insipid flavor, often speckled with red or misshapen, with red streaks in the flesh. In the natural vector of yellows, the plum leaf hopper, *Macropsis trimaculata*, the virus has an incubation period of from 8 to 26 days. The virus cannot be transmitted by mechanical means.

Little peach (*Chlorogenus persicae* var. *micropersica*) is found in the same area as yellows and its causal virus is a strain of the yellows virus, despite very different symptoms. Trees with little peach have foliage that at first looks quite healthy, but later becomes yellowish, and twig growth is slow, stunted, and bushy. No wiry shoots occur as in yellows. The fruits, which ripen about 10 days later than on healthy trees, are undersized with insipid flavor. The trees gradually decline and die in about four years. Transmission of little peach is the same as in peach yellows.

Phony peach (*Nanus mirabilis*) is a disease of the southern states. Affected trees have abundant dark green, dense foliage, so that they may appear to be the most vigorous trees in the orchard, but their growth is retarded, and although they may live for many years, they produce small fruit of normal color but undesirable flavor. Nectarine, almond, apricot, and plum also are susceptible. The disease can be identified by a chemical



FIG. 170. Peach mosaic. (*Top*) Affected tree in early spring; note the retarded foliage as compared with healthy trees in the background. (*Bottom*) Symptoms of mosaic mottling, stunted development, and distortion in leaves of affected trees; compare with healthy leaf at right. (Courtesy, E. W. Bodine, Col. Agr. Exp. Sta.)

reaction of the wood of the roots which shows purple spots when treated with acidified methyl alcohol. The above-ground parts of the tree do not appear to contain virus. For this reason the disease is not spread by top grafting or budding, but by root grafting. No insect vector is known. The virus can be removed from the roots by a heat treatment.

Peach mosaic (*Marmor persicae*) is prevalent in the southwestern states where it is gradually being brought under control by a Federal and State eradication program. It is a disease difficult to diagnose because the reliable symptoms are transient in the spring. These consist of variegation of the blossoms and yellowish mosaic and puckering of the veins (Fig. 170). The internodes are irregularly short and long, with an abnormal number of short side spurs or branching of the twig tips which gives the tree a tufted appearance. The fruit is bumpy, often late in ripening, with no constant discoloration and more or less normal flesh.

There are several strains of the peach mosaic virus producing different symptoms on peach. The virus is carried without symptoms in varieties of plum, prune, apricot, and almond, making these undetectable sources of infection. The disease can be transmitted by budding or grafting, and by the green peach aphid, *Myzus persicae*.

Peach X-disease or yellow-red virosis (*Marmor lacerans*) is a rapidly spreading, dangerous disease prevalent in the northern states, with a counterpart, the Western X disease, in the Pacific Northwest. Affected peach trees leaf out as though normal, but later develop on the leaves yellow patches and red spots which fall out, giving the leaf a ragged appearance. Soon all leaves, except those at the twig tips, fall. The fruits shrivel and fall soon after the leaf symptoms appear or remain as mummies on the tree, or in milder cases they ripen prematurely and are bitter and without an embryo in the pit. Often only part of the tree is affected. Diseased trees fail to produce a good crop year after year, but rarely are killed by the virus.

The principal wild host is the chokecherry, *Prunus virginiana*, from which orchards become affected. Diseased chokecherries have similar symptoms, but the foliage is brilliantly reddened so that the affected trees are easily recognized at a distance. The nectarine also is attacked.

The X-disease is not believed to spread naturally from peach to peach, but only from chokecherry to peach, which reduces control to a campaign of chokecherry eradication. The southern and southwestern states may be safe from this disease, since the chokecherry does not occur in these regions. The virus can be transmitted by budding. No insect vector is known.

Control of Stone Fruit Viruses. The same basic control measures apply to all these and other stone fruit viruses. They include locating and

destroying diseased fruit trees and wild stone fruit hosts near the orchard, and prevention of spread of the diseases in nursery stock by use of disease-free propagation wood and quarantine laws which control the movement of stone fruit nursery stock out of infested areas.

Location and eradication of diseased trees is carried out jointly by the States and the Federal Bureau of Entomology and Plant Quarantine. In the case of chokecherry, eradication of trees within 500 ft. of the orchard, using an herbicide such as sodium chlorate or ammonium sulphamate, is advised. Laws require the removal of diseased trees and prohibit or supervise the movement of nursery stock from infested areas to noninfested ones. Enforcement of these laws has been difficult at times, particularly because of the problem in diagnosing these diseases. The program has been useful however, in restricting the infested areas, and in some cases in completely eradicating the disease from infested counties. Several states have recently adopted programs for the certification of virus-free budwood. There is little promise in control of these diseases by breeding because of a lack of resistant types to serve as breeding parents. Peach trees affected with X-disease have been cured by injections of *p*-aminobenzenesulfanilamide, but it is not known whether this will have practical importance.

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Chapter 12

Diseases Caused by Parasitic Seed Plants and Algae; Epiphytes

Parasitic Seed Plants

While the great majority of flowering plants are autophytes, manufacturing their foodstuffs by photosynthesis from carbon dioxide and the minerals and water that they secure from the soil, a few of them have other means of obtaining their nutritional essentials. Among the higher plants there are colorless ones that derive their sustenance saprophytically from dead organic matter, others that supplement their diet with artfully caught insects, as the sundew and pitcher plant. There are seed plants that derive benefit from mutualistic association with other organisms, as the nodule-bearing legumes, and finally there are those that steal part or all of their sustenance from other plants. It is with these last, the partially or entirely parasitic seed plants, that we are here concerned.

These seed plant parasites differ in the extent and manner of their parasitism. They may be entirely dependent on a host, or only partially so, and they may parasitize either stems or roots. With the exception of the broomrapes, which at times are destructive to tobacco, hemp, and clovers, the root parasites are not highly injurious to their hosts. One of the root parasites, the sandalwood tree, is highly prized for its wood. In contrast, the stem parasites, mistletoe and dodder, are exceedingly injurious at times.

DODDER (*Cuscuta spp.*)

History and Distribution. There are more than 160 species of dodder, some 40 of which are found in the United States. They are known under various descriptive names, such as strangleweed, love vine, gold thread, pulldown, clover-silk, devil's hair, and others. The dodders are worldwide in distribution but best known as pests in Europe and the United States. Linnaeus recognized the dodder as a parasite and undoubtedly its noxious effects had been observed centuries before. Recent work on dodder has dealt mainly with the physiologic and morphologic relationship between dodder and its host, the control of the pest through seed legislation and cultural practices, the development of ingenious means for

freeing crop seed from dodder seed and the use of dodder bridges in transferring viruses from one host plant to another.

Importance. Losses from dodder have been most serious in legumes, such as clovers and alfalfa, and in flax. In parts of Europe, clover growing has been abandoned and flax production cut in half because of the destructiveness of dodder, but conditions in the United States seem to be less favorable to dodder injury. Nevertheless, there are frequent reports of serious local losses from dodder, especially in alfalfa and clover seed-producing states. These losses are aggravated by extremely strict legislation which in some states condemns seed that is contaminated with dodder seed. Occasionally vegetables, ornamentals, young nursery stock, and even nursery trees may suffer appreciable losses from dodder.

Symptoms and Signs. Dodder may be recognized from a distance as yellow or orange patches in otherwise green fields. The yellow mass is a dense tangle of thin, wiry stems overgrowing the normal vegetation. (Fig. 172.) In advanced cases, the spot is in the form of a large yellow ring of dodder, 30 feet or more in diameter, surrounding a black, burned-over-



FIG. 171. Dodder on clover showing the habit of the parasite and its blossoms. Note the devitalized appearance of the clover plants in the center of the photograph. Soon they would die. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)



FIG. 172. A field of lespedeza completely overgrown with dodder. Such heavy infestation may result from planting "bargain lots" of seed which are cleanings or waste from the seed-cleaning process. (Courtesy, Ala. Dep. Agr.)

appearing center where the host plants have been entirely destroyed. In very severe cases, entire fields may be overgrown with the pest.

On examining the dodder closely, tiny scales or rudimentary leaves are seen on the stems, and as the plant matures, dense clusters of tiny, pale blossoms appear (Fig. 171). These are found especially along the stems of the host plant. Large quantities of tiny gray or reddish-brown seed are produced, as many as 3,000 from a single plant. While the seed somewhat resemble that of the small-seeded legumes, they can be distinguished by the fact that they are roughened, with 3 flattened sides, and do not have a conspicuous scar.

Plants attacked by dodder show symptoms of starvation, stunting, and pallor, and they soon die. Since the dodder is an obligate parasite it dies with the host unless new host plants are available. Ordinarily, the dodder stems extend outward, grasping new hosts as the old ones fail, and thus steadily enlarging the spot of infestation.

Etiology. Several species of *Cuscuta* are dodders of practical importance in the United States, particularly *C. campestris* the field dodder, *C. gronovii* the common dodder, *C. epithymum* the clover dodder, *C. indecora* the large-seeded alfalfa dodder, and *C. approximata* var. *urceolata* which is quite damaging in the western states. The botanical features of these and other dodders are given by Hansen, and Yuncker, cited in the references at

the end of the chapter. Although the dodder is a true seed plant, it exhibits remarkable adaptations to its parasitic mode of life. The seed will mature even if the host is destroyed before the seed has normally matured. It will retain its viability for several years. In the spring the seed is adapted to germinate a little later than the host seed so that host plants are available to the dodder seedling when it appears. In germination, the dodder seed sprouts to form a hairlike shoot (Fig. 173). This anchors itself in the soil and the free end of the leafless seedling rotates until it comes in contact

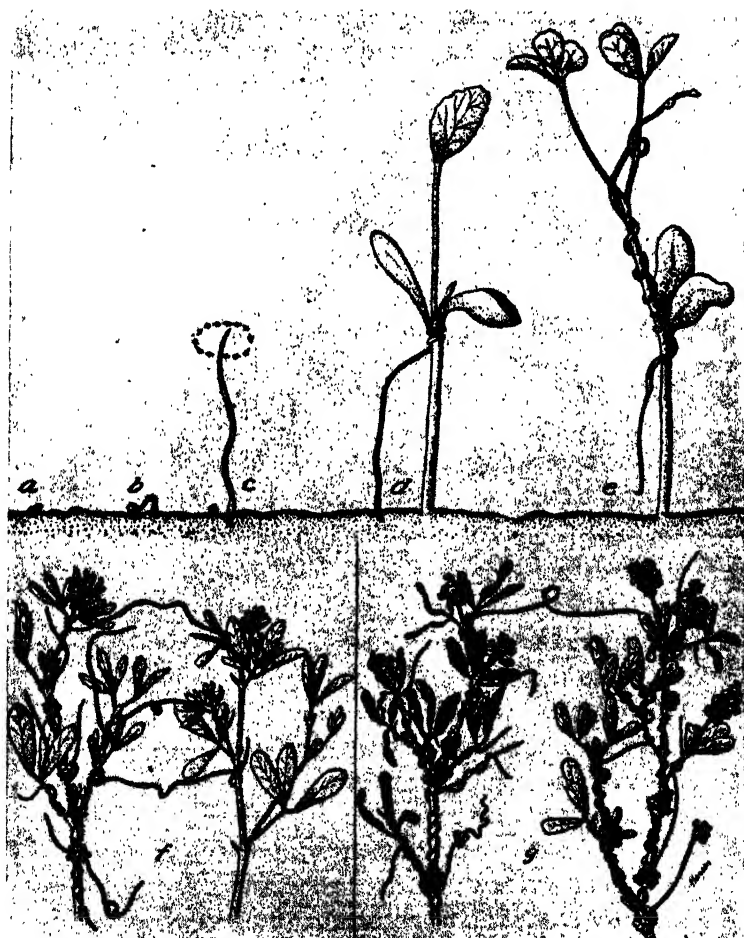


FIG. 173. The life cycle of dodder. (a, b) Germination of the seed. (c) Rotational movement in search of a host plant. (d, e) Feeding contact has been made and the dodder stem has lost contact with the soil. (f) Lateral spread of the dodder stems from plant to plant. (g) Blossoming and seed production of the parasite. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

with a stem or other foreign object. It then makes two or more coils about the foreign object. If the latter is not a suitable host (e.g. a straw) the free end continues rotating, and when contact is made with a susceptible stem, there are formed on the inner, contact surfaces of the coils, little toothlike swellings or haustoria, which force their way into the host stem, connect with the xylem and phloem elements of the stem, and begin using the water and food materials of the host plant. The lower end of the dodder stem dies away, so that the parasite loses all contact with the soil. If contact is not made with a suitable host, the seedling dodder stem may remain alive for several weeks, but then dies. Once the nutritive relation is established, the free ends of the dodder stems continue to make new host attachments until great masses of the parasite result. If the dodder stems are broken off and scattered, as in haying operations, the stem pieces may attach themselves to new hosts, thus acting as vegetative organs of reproduction.

Normally, the dodder overwinters as seed, either in the soil, or mixed with crop seed. A few species, however, notably the clover dodder, are perennial, and these may rarely produce seed. While the first introduction of dodder into a field commonly is due to the use of contaminated seed, other means of infestation are dodder fragments or seed from hay, irrigation water, accidental spread by men or animals, and infested manure. The seeds will pass uninjured through the digestive tract of animals, which increases the danger from this source.

Epiphytology. The wide geographic distribution of dodder, its constant appearance year after year in the same fields, and its prevalence on roadside weeds under most unfavorable growing conditions, attest the fact that dodder tolerates a wide range of environmental conditions. It has many hosts and will flourish under almost any conditions that permit the host plants to develop. With its various adaptations for spread and survival, dodder can be regarded as a highly successful parasite under natural conditions, and this parasitic efficiency is further aided by man in local dissemination through agricultural operations and in long-distance spread by contaminated crop seed.

Control: 1. PREVENTING INTRODUCTION OF DODDER-INFESTED SEED. As agricultural seed is the main original source of infestation, avoidance of dodder-infested seed, by regulation or by careful examination of seed, stands foremost among the control practices. It has been said that dodder has been more legislated against than any other weed, possibly excepting the Canada thistle. In parts of Europe it is unlawful to sell crop seed containing even a single seed of dodder. Federal law excludes from the United States all commercial seed containing one or more dodder seed

per 5-g. sample. Many states have laws strictly regulating the sale of dodder-infested seed, and other states require a statement on the seed-sack label indicating the presence or amount of noxious weed seed, including dodder. Even with this supervision, however, a certain amount of dodder-infested seed is always to be found on the market. For this reason the buyer should examine his seed, especially that of small-seeded legumes, for the rough, flat-sided dodder seed, and if any doubt exists, the seed should be inspected by the state seed laboratory for the presence of dodder. Seed, even with small amounts of dodder contamination should be rejected.

2. PREVENTING INTRODUCTION IN HAY AND MANURE. If hay and manure are secured locally, it will be possible to investigate the source and reject them if there is danger of dodder contamination. It may be difficult to detect dodder in commercial hay, but the grower should be aware of the danger of dodder in such hay, and guard against introduction of the pest by this means.

3. CLEANING DODDER-INFESTED SEED. If there is a marked difference in size between the dodder seed and the crop seed, the former may be removed by screening, as in the case of small-seeded dodder in clover or alfalfa seed. The recommended screen has 20 meshes to the inch of No. 30 to 34 wire. In most cases it is not advisable to attempt to clean such seed if noninfested seed is available, and this is true especially of large-seeded dodder in small-seeded legumes.

The commercial seed industry has worked out efficient methods of cleaning dodder-infested crop seed, including an ingenious device by which crop seed is mixed with iron powder. Much of the powder sticks to the rough dodder seed which is then drawn out of the crop seed with magnets.

4. KILLING DODDER IN INFESTED SEED. A method for killing dodder seed by dry heat, without injuring the crop seed, has been developed in Europe and appears promising, but as yet this has not been adopted in America.

5. SELECTING DODDER-FREE SITES FOR CROP PRODUCTION. It must be remembered that dodder is a common pest on weeds and other wild plants. Before breaking ground for a crop on native sod, the field should be examined for presence of dodder. If found, it should be eradicated by the means given below before the land is used for a susceptible crop.

6. ERADICATING DODDER IN CASES OF LIGHT INFESTATION. Where only a small amount of dodder is present in a field, mow the dodder areas before seed is formed, allow the mowed plants to dry, or wet them with crude oil or kerosene and burn them, or burn them over with a blow torch, or feed the cut plants for hay, being careful not to distribute the pest in the

process. If seed has formed, burn over the infested spots. For a few weeks the burned spots should be repeatedly but lightly hoed to allow buried seed to germinate and die. The treated spots should be examined frequently to make sure that the eradication is complete.

Eradication of small spots of dodder by chemicals is practiced sometimes. This has not been generally recommended in the past because of the cost, inconvenience, and danger in using the chemicals. The recent development of new herbicides, however, offers new possibilities of dodder eradication by chemicals. In Peru, where dodder on flax is important, successful eradication has been accomplished by careful spraying with sodium hydroxide, 1-100.

7. ERADICATING GENERAL INFESTATIONS OF DODDER. Large areas of dodder in a field usually result from negligence in the past. Where such infestations exist, the field should be mowed before seed has formed, and the hay fed to stock, preferably in the field. The land may then be plowed and reseeded or closely grazed, to prevent formation of late seed. In early stages, close grazing alone, as by sheep, may suffice to prevent seeding of the dodder.

If the dodder has matured seed over large areas, the problem is very difficult, because the dodder seed will retain viability in the soil for five years or more, and susceptible crops cannot be used on the land for that period. Soybeans, cowpeas, or velvet beans might be substituted, as these are rarely attacked by dodder. In extreme cases the entire field may be mowed, allowed to dry, and then burned over. This should be followed by fallowing for the remainder of the season, then beginning a five-year rotation starting with a cultivated crop such as corn or sorghum, and then planting dodder-immune crops the remaining four years. The heavily infested crop could be cut for hay, stacked, and fed in the field, but this leaves the danger of spread to other parts of the farm. It goes without saying that a dodder-infested crop should not be harvested for seed.

MISTLETOE (*Phoradendron* spp. and *Arceuthobium* spp.)

History and Distribution. In America, two genera of mistletoe are found, the leafy mistletoes, species of *Phoradendron*, which are common in the southern states and occur principally as parasites on broad-leaved trees, and the dwarf mistletoes, species of *Arceuthobium* which are restricted to conifers, for the most part in northern forests. There are about 80 species of *Phoradendron*, all in the Americas. *Arceuthobium* comprises five species, and several physiologic forms, all found in the northern hemisphere, but not in Europe. The classic mistletoe of Europe is *Viscum album* which consists of three physiologic forms, one occurring on hard-

woods, one on fir, and one on pine and spruce. While the mistletoes have been known for many centuries, their recognition as destructive parasites and the study of their pathology is comparatively recent, based largely on the extensive studies of von Tubeuf in Europe (1923) and of Bray on the leafy mistletoe (1910) and Weir on the dwarf mistletoe (1915 ff.) in America.

Importance. Under many conditions the leafy mistletoes are relatively harmless, but in the southwestern states where tree growth is not vigorous, shade and forest trees may be seriously handicapped, and hackberries and oaks are often killed by the pest, while incense cedar stands may be markedly retarded in growth. More serious damage is sustained by the northern softwoods from the dwarf mistletoes, which are rated next in importance to the wood rots as causes of loss in these forests. Here the infestation results in heavy mortality or serious retardation of growth even in relatively young trees. In addition, mistletoe-infested trees are predisposed to attack by bark beetles. Even in cases where the tree growth is not seriously checked, the value of lumber from mistletoe-infested trees may be very low, owing to wood faults produced by the mistletoe haustoria and to abnormal branching. For example, in the Bitterroot and Missoula valleys of Montana there are great areas where the important Douglas fir is so badly injured by mistletoe that this tree is not even included in estimates of forest resources of these areas.

Host Plants. The great majority of tree species are attacked by one or more of the mistletoe species. The leafy mistletoes are found particularly on oak, elm, maple, sycamore, the gums, mesquite, Bois d'arc, sugarberry, walnut, ash, poplar, willow, buckeye, incense cedar, and more rarely on fir, juniper, and cypress. The dwarf mistletoes are most serious on pines, firs, larch, hemlock, spruce, and tamarack.

Symptoms and Signs. The mistletoe plant itself is an obvious and unmistakable indication of infestation (Fig. 174). The plant of the leafy mistletoes is evergreen and rather large and dense, up to several feet in



FIG. 174. Heavy infestation of broadleaf mistletoe on black gum. Louisiana, 1932. (Photograph, A. F. Verrall.)



FIG. 175. Dwarf mistletoe (*Arceuthobium campylopodium* f. *divaricatum*) on single-leaf piñon pine, California, showing immature flowers and fruits. Little swelling of the limb is seen, although frequently the dwarf mistletoes produce large, musclelike swellings. (Courtesy, L. S. Gill.)

diameter, occurring on the upper branches of the host tree. In winter, these dense tufts of mistletoe can be recognized at long distances. Often the tree branch is swollen or distorted at the point of attachment of the mistletoe stem. The plant is a yellowish or olive green, usually with broad, thick, leathery, oval leaves with spikes of inconspicuous, apetalous flowers and, later, berries, which usually are white with a sticky mesocarp.

A cross section of the host branch through the point of mistletoe attachment shows rootlike feeding organs (haustoria) penetrating the wood to various depths. The haustoria consist largely of xylem vessels which are attached to the xylem elements of the host branch, enabling the mistletoe to make free use of the water and mineral salt supply of the host tree.

Shoots of the dwarf mistletoe are in dense clusters but only a few inches long (Fig. 175). The leaves are reduced to inconspicuous scales, and the shoots are a dull yellowish or greenish brown in color. The blossoms are inconspicuous, occur in spikes, and produce abundant

berries, each containing one or two seeds embedded in a sticky pulp. The attacked branch usually is swollen, and abnormal or excessive branching may be induced so as to result in dense brooms, sometimes involving all or most of the tree. Haustoria extend into the wood as with the leafy mistletoes.

Heavily infested trees show symptoms of impaired vigor with chlorosis, reduced size and number of leaves, dead or broken branches, and die prematurely.

Etiology and Epiphytology. The mistletoes are disseminated solely by their seeds. Birds are the chief vectors involved, although washing of the seeds by rain aids in local spread, and in the case of the dwarf mistletoes the seeds are shot out violently to a maximum horizontal distance of 33 feet. The adhesive substance on the seeds enables them to adhere to branches and also favors their being deposited on branches by birds in the act of wiping them off the bill.

The seeds germinate readily, the radicle forming a disk-shaped appressorium that adheres tightly to the bark, in the center of which a peg-like haustorium penetrates through to the cambium. Only relatively young, thin bark can be thus penetrated, and generally the haustorium makes use of lenticels or buds in its ingress. By modification of the meristematic tissues of the haustorium and those of the tree cambium, the newly developed xylem of the tree becomes continuous with that of the parasite, and the water and salts of the tree become available to the mistletoe. This is perhaps favored by the fact that the mistletoes have a higher osmotic pressure of their cell sap than is true of their hosts, thus leading to a water exchange in favor of the parasite. Branches of the haustorium also extend up and down and about the tree following the cambium layer, and here and there produce secondary haustoria. The haustoria do not penetrate into the wood, but as the tree grows and adds annual rings of wood, the haustorium elongates outward, thus becoming buried in the tree. From the number of rings between the tip of the primary haustorium and the bark of the tree it is possible to tell the age of any mistletoe infection, and this may be from 60 to 70 years or more. Two specimens have been reported 219 and 419 years old respectively.

In contrast to dodder, the mistletoes are hemiparasites or partial parasites. Although the host plant furnishes water and mineral salts, the mistletoe contains chlorophyll and manufactures its own food. It has even been claimed in Europe that when a tree is defoliated, by insects for example, some of the food elaborated by the parasite is furnished to the tree. Pollination of the mistletoe is accomplished by insects.

As well as being parasites, the mistletoes in turn are attacked by a

number of insects and fungi, the latter including one or more species of rusts on the leafy mistletoes and a fungus (*Wallrothiella arceuthobii*) that destroys the immature fruits and sometimes kills the plants of the dwarf mistletoes.

Control: 1. IN ORNAMENTAL AND SHADE TREES affected by the leafy mistletoe the pest can be held in check by pruning out the parasitic growths as they appear. For complete eradication it is necessary to cut off the affected branch several inches below the mistletoe, but a fairly satisfactory practice is merely to pull off the aerial portions of the parasite with a curved mistletoe hook, although this does not destroy the haustoria in the wood. In some cases there is opposition to mistletoe eradication because of traditional sentiment or desired decorative effect of the brooms.

2. IN FOREST TREES mistletoe, especially the dwarf mistletoe of northern forests, is not easily dealt with. It is a problem associated with open, mature or overmature stands, and in the past it has been difficult to dispose of these stands. The best solution lies in felling affected trees, as this destroys the obligate parasite. Lumbering contracts should require the felling of all infested trees whether useful for lumber or not. In some cases this will require clean cutting, in which event measures must be taken to assure regeneration of the forest, as by delaying the cutting of a few seed trees until the new stand is established. Such cutting is best accomplished in late summer before the mistletoe fruits ripen but when a good vegetative growth makes it easy to recognize affected trees. Particular attention should be given to avoid bringing mistletoe into uninfested regions on seedling trees, since many of the species are restricted geographically. With the established fact of physiologic specialization within this group and the frequent observation that individual trees will be mistletoe-free although surrounded by heavily infested trees, there is reason to believe that selection of tree strains for mistletoe resistance offers good possibilities for the future.

WITCHWEED (*Striga lutea*)

This foreign flowering plant parasite is discussed briefly because it is an outstanding example of control of such a parasite by breeding. The witchweed is a pest causing much damage to sorghums and other grasses in Burma and to rice in the Dutch East Indies. With the exception of the stalk borer it is responsible for greater loss in corn and sorghum in South Africa than all other diseases and pests combined.

Striga lutea is a root parasite, its roots forming haustoria which penetrate the roots of the host plant from which it derives water and food, eventually wasting and destroying the host. Seeds of the parasite germi-

nate only in the presence of a substance secreted by the host roots and are conserved until a host is available. After a period of root feeding the stems of the parasite come up through the soil and produce reddish-yellow flowers and great numbers of seed that return to the soil.

Control of witchweed is aided by clean cultivation, use of trap crops, wide spacing of plants, and spraying the weed with herbicides. Resistant native sorghum strains of poor agronomic value have been crossed with commercial sorghums to produce desirable resistant types.

Parasitic Algae

The algae, most of which are autophytic, are a group of very minor importance in pathology. Less than 1 per cent of Sorauer's extensive handbook on parasitic plant diseases is concerned with those caused by algae. Yet we find here at least one disease of considerable economic importance and several interesting instances of parasitism by algae in noneconomic plants.

Two or more of the green algae, in particular *Cephaleuros virescens* and *C. parasitica* cause serious injury in tea and coffee plantations, a disease known as red rust or orange rust. The former species also infects a number of other tropical or southern plants, including citrus fruits, mango, jujube, persimmon, guava, acacia, pecan, magnolia, jasmine, fringe tree, privet, viburnum, and rhododendron. The disease may occasionally give trouble in greenhouses. It is spread by airborne sporangia which germinate to produce zoöspores in drops of water. These enter through stomata and form myceliumlike chains of algal cells that grow through the host tissues, producing extensive dead spots on the surface of which are borne reddish hairs, some of which are tipped with clusters of zoösporangia. Host leaves are spotted and branches and twigs are girdled, stunted, and made more susceptible to attack by fungus diseases.

Epiphytes

A number of plants including flowering plants, fungi, lichens, and algae, customarily grow over the surfaces of other plants not parasitizing or deriving any benefit from the supporting plant except mechanical support. Occasionally these epiphytes may be harmful by shutting off light or gas exchange, breaking branches by their weight, or even strangling the supporting plant as in the strangler fig tree and the climbing bittersweet. Grapevines have been found responsible for suppression or even death of trees. The Spanish moss (*Tillandsia usneoides*), which resembles a lichen but in reality is a flowering plant of the pineapple family, at times has a repressing or killing effect on its supporting plants by excluding light and

smothering. The smothering effect of the slime mold, *Mucilago spongiosa*, was pointed out on p. 237.

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Chapter 13

Diseases Caused by Nematodes or Eelworms

Many nematodes (nemas, eelworms, or roundworms) are saprophytes in soil and water, others are predatory on one another, some are important intestinal parasites of higher animals, as the roundworms of horses and swine and the hookworm of man, a number are parasitic on plants including a few species that are causes of important diseases of economic crops. The free-living species or those parasitic on plants usually are microscopic; the animal parasites include some forms that are larger, sometimes up to several inches in length.

ROOT KNOT (*Heterodera marioni*)

Distribution and Importance. Root knot is a common and serious disease in all warmer climates and in greenhouses everywhere. Because of its wide host range it ranks as one of the most important diseases of agriculture. Few southern garden soils are free of the pest. The disease is of economic importance to the grower of nursery stock because even lightly infested stock cannot be sold and often is a total loss. The pest is so widespread in the South that it appears almost invariably in plantings of susceptible crops in light soils after a few years of continuous cropping. In certain crops the loss is increased because root knot predisposes the plants to injury by other diseases. Thus wilt-resistant varieties of cotton, tomato, and watermelon become susceptible to wilt if the root knot organism is present.

Host Plants. The root knot nematode attacks more than 1400 species of plants. The list of the more important plants attacked includes alfalfa, beans, beets, cabbages, clover, cotton, most cowpeas, cucumbers, elm trees, Korean lespedeza, muskmelons, okra, peach trees, peas, pecans, roses, most soybeans, spinach, strawberries, tomatoes, vetch, watermelons, and many other field, garden, ornamental, and fruit crops, as well as numerous weeds. A number of important crops are resistant to the disease. These include nearly all grain and grass crops except rice, cowpeas of the varieties Iron, Monetta, Brabham, Victor, Resistant Texas Blackeye No. 8152, and Conch, soybeans of the varieties Armredo,

Laredo, Biloxi, and O-too-tan, velvet beans, sericea lespedeza, cotton varieties Acala, Early Wilt, 4-in-1 strains 3 and 4, Hi-Bred, Cleveland (Wannamaker), and Rhyne's Cook, peanuts, crotalaria, bur clover, some varieties of peaches, plums, apricots, and cherries, resistant strains of beans, lima beans, and lettuce, and numerous species of ornamental plants. The bush fruits are not highly susceptible. Vegetables that are affected but yield profitable crops even when attacked include asparagus, cabbage, cauliflower, celery, horse radish, onion, parsnip, radish, rhubarb, spinach, turnips, and numerous sweet potato varieties. Susceptible legumes escape the disease if grown as winter crops, and susceptible vegetables can be safely grown as early spring crops before the nematodes are active. Many weeds are susceptible and are useful as indicators of root knot in the soil but are harmful to the extent that they maintain the nematode population in the soil.

It is very important to bear in mind that many crops which show enough resistance or tolerance to permit fairly normal growth, still allow the nematodes to reproduce, and are therefore of little value in crop rotations designed to starve out the nematodes. This includes corn, sweet potatoes, the soybean and cowpea varieties listed above, and some grasses, such as Bermuda and species of *Paspalum*. More extensive lists of susceptible and resistant plants are given in references by Godfrey and Tyler, cited at the end of the chapter.

Symptoms and Signs. Affected plants exhibit symptoms of severe water deficiency, are stunted with wilting, yellowing, and ultimate death. The roots have many small and large swellings involving the entire thickness of the root in contrast to the beneficial legume nodules which are attached at the side of the root (Fig. 176). The only other swellings commonly found on roots are club root of the crucifer family (Fig. 124, which usually lacks the numerous small swellings, and crown gall of woody plants, with one or several large, woody knots at the side of stems or roots, usually at the soil line (Fig. 150). Within the root knots are pear-shaped, adult female nematodes just visible to the unaided eye. In advanced stages the knots show extensive decay caused by secondary fungi and bacteria attacking the weakened tissues.

Etiology. When mature, the female nematode extrudes a jellylike sack filled with 500 to 3000 eggs (Fig. 177). Within the older eggs, the wormlike larvae can be seen with the aid of a microscope. The larvae hatch, escape from the decaying knot, move about a short distance in the soil, and attack the root at a new point, imbedding themselves in the root tissues, and secreting a stimulating chemical which causes a new knot to form around each larva. Fertilization of the females may occur or may be

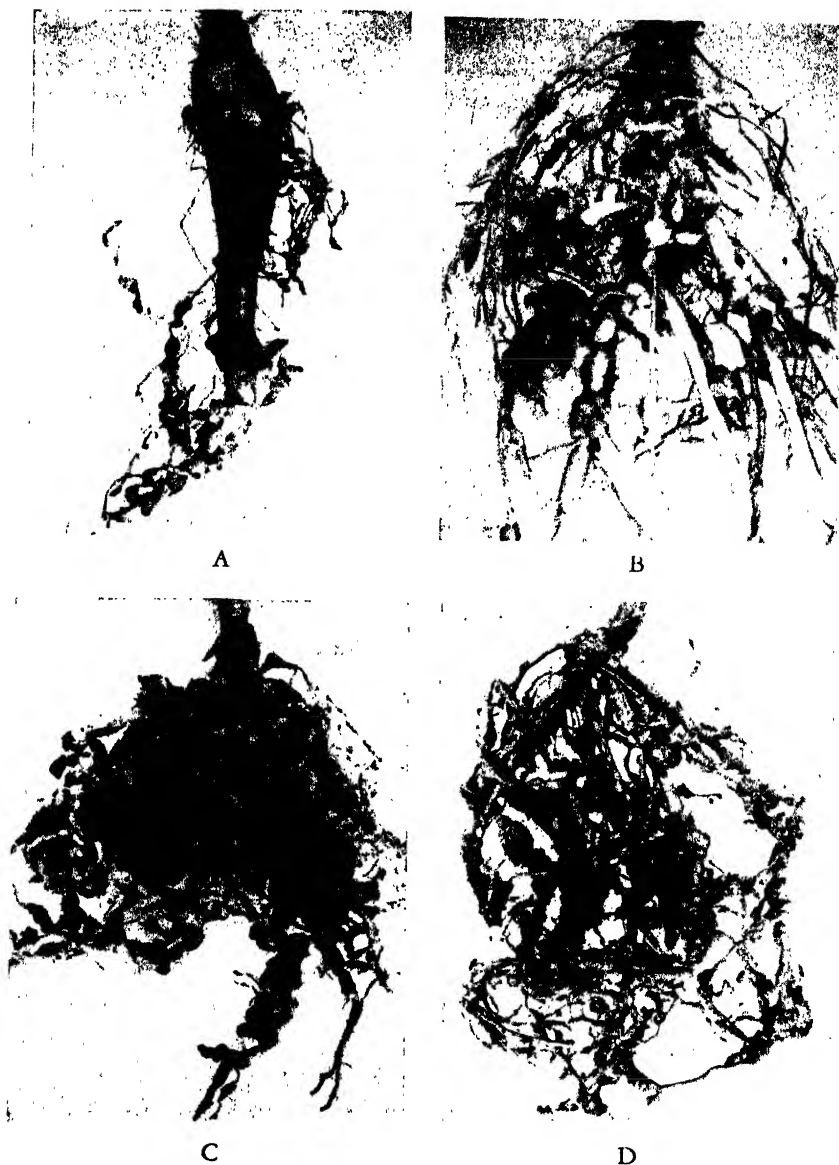


FIG. 176. Root knot. (A) On carrot. (B) On mung bean. (C) On snapdragon. (D) On peach.

omitted. Under optimum conditions the life history is completed in from 25 to 30 days, but this is lengthened to as much as 80 days in cool climates. The nematode exhibits physiologic specialization, discussed on p. 366.

Epiphytology. By their own efforts the nematodes move only very short distances—not over a foot in a month. They are spread rapidly down

furrows with surface or irrigation water. Any manner of moving soil, except windblown dust, will spread the nematodes across a field or from one field to another, as on farm machinery, shoes, or hooves. They are not carried in seed, but often are introduced into vegetable plantings on the roots of infected tomato, cabbage, strawberry, lettuce, and sweet potato plants, or on Irish potato tubers, and in the same way they can be introduced with diseased nursery stock.

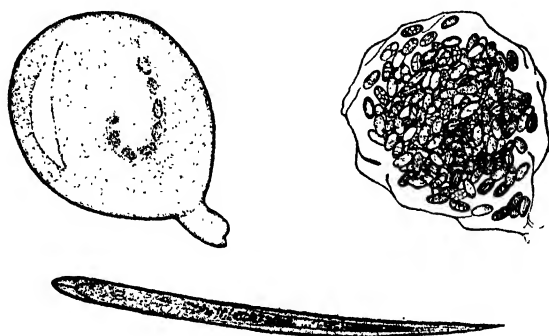


FIG. 177. Root knot nematode. (*Top, left*) Immature female. (*Top, right*) Egg sack containing eggs. (*Bottom*) Freshly emerged larva.

High temperatures favor nematode development which occurs at soil temperatures from 58° to 89° and is most active at 81°F. The worms are most prevalent in the southern states and in greenhouses, where the long period of warmth increases the number of generations. In Oklahoma there are six or seven generations a year, in Florida twelve, and farther north only one or two. The nematodes are most abundant in light sandy soils which aid free movement. Most of them are at the depth of the root zone, from 3 to 10 in. deep, but in association with deep-rooted plants they may be found at a depth of 6 ft. or more. Their spread is favored by temporary water transport, but they are destroyed by prolonged flooding. The worms and eggs are killed by temperatures of 118°F. in 30 minutes. This amount of heating to a depth of one to several inches occurs naturally in summer in some southern soils. Soil moisture has little effect on the nemas except that they are not favored by extremes of wetness or dryness. The nemas themselves and their eggs soon die if completely dried out. Larvae are killed in 20 to 25 minutes and eggs in masses in 5 hours if exposed to sunlight. A high percentage of organic matter in the soil reduces nematode populations by favoring the growth of predaceous nemas and mites and of fungi which destroy the nemas. Use is made of this in nematode control. Heavy fertilization also is helpful in stimulation of vigorous root

development of affected plants so that they are able to grow more or less normally in spite of some nematode root damage.

Control. Control of root knot may be accomplished by different practices, depending on how the crop is grown. Except under greenhouse and seedbed conditions, complete and permanent eradication of the parasites is not practicable. The most that can be accomplished is to reduce the worm population to a low level and keep it there. This may be done in the following ways.

1. **IN FIELD CROPS.** Crop rotation in which susceptible crops are grown once in two or three years is a standard control practice. Immune or highly resistant crops such as small grains, sorghum, crotalaria, or velvet beans, should be used in the rotation. Susceptible weeds should not be allowed on the land, nor should susceptible legumes be interplanted with the resistant crop. As much organic matter as possible should be returned to the land, in the form of green manure crops or manure.

In hot climates, land can to a great extent be freed of the nemas by three successive midsummer plowings, 3, 6, and 9 in. deep. Summer fallow with occasional cultivation is helpful in reducing nematode populations. In tobacco, root knot can be reduced to a minimum by plowing up the roots immediately after harvest, to expose them to the sun and drying, and maintaining clean fallow till fall when a green manure crop, such as oats, can be grown and plowed under. Crotalaria may be grown on the land instead of fallowing it.

Soil fumigation, as described below, is not recommended on a field basis except with crops of high acre value and with widely spaced crops, such as watermelons, where spot disinfestation of the planting points with a fumigant is practical. Where water is available, flooding the land for a month or more will aid in control. Sometimes good results are obtained by using a trap crop, such as susceptible legumes. If the nematodes are allowed to infest the crop, and it is plowed under before the nemas mature, they will be killed and the population of nematodes in the soil greatly reduced. The plowing must be carefully timed, for if delayed, an increase in nematode population will result.

In nematode-infested soil preference should be given to the moderately resistant varieties of susceptible crops, such as the varieties of cowpeas, soybeans, and cotton listed above. There are as yet no resistant commercial tobacco varieties, but resistant breeding stocks have been found and are being used in development of resistant commercial varieties.

2. **IN FRUIT ORCHARDS.** In starting a new orchard, land which has not been planted to susceptible crops or overgrown with weeds for a number of years should be selected if possible. The condition of the land, with

reference to root knot, can be tested by examining the roots of susceptible weeds which may be present. The common lamb's quarters and the wild lettuce are particularly good indicators of root knot. Or some of the soil may be planted with seed of a susceptible crop such as okra, vetch, cowpeas, or tomatoes. If these show no knots after four to six weeks, the soil can be considered relatively free from the parasite. The young trees should be carefully inspected for root knot before planting, and no knotted trees should be accepted or set out. Insofar as possible, root knot-resistant varieties and rootstocks should be used, and detailed lists of these are available at the state experiment stations. Where a cover crop is desired, it is best to use root knot-resistant crops. Trap crops may be used as indicated for field crops. Where only a small part of the orchard is infested, the trees may be removed and destroyed, and the soil in their vicinity sterilized by one of the methods described below. Applications of urea are helpful. If the orchard is badly infested, production can continue if the trees are stimulated by liberal fertilization and cultivation, but in such cases it may be best to start a new orchard site with a view to abandoning the old site as soon as the new one is producing.

3. IN NURSERIES. Most of the suggestions above on controlling root knot in the orchard also apply to the nursery. A considerable number of varieties of nursery stock and ornamentals are root knot-resistant, and full use should be made of these in nurseries subject to this disease. In case part of the nursery is infested it may be a good plan to treat the land as suggested for field crops before replanting with susceptible stock, and some of our nurserymen do this as a regular practice. Most of the evergreens are resistant to root knot and may be successfully grown in infested land. Chemical methods of soil disinfestation as described below can and should form a part of the routine in the greenhouse and beds, and the expense of this will sometimes be justified in the nursery.

It has been found that heating at 118°F. for 30 minutes destroys the root knot parasite in infested roots, and some nursery stock can withstand this temperature without injury. Where nursery stock is only lightly infested with root knot (up to 5 per cent) the hot water treatment is permissible as a means of disinfesting stock. The treatment consists in immersing the roots in water for one-half hour, usually at 118°F. The work may be done in a vat, controlling the temperature by means of steam which bubbles through the water in the vat at a controlled rate. This temperature does not injure some types of ornamentals (black locust, peonies, and tuberoses), but growers are advised not to use the method on other plants except in an experimental way until definite data are available on the resistance of the various species to this temperature.

In their attempts to control root knot, the nurseryman and the ultimate grower are aided by the conscientious efforts of the state nursery inspectors to keep badly infested stock out of the trade. Most states have no specific law on root knot-infested stock, but interpret the clause on unsaleable nursery plants to include stock more than 5 per cent of which is obviously infested, while lighter infestations are heat-treated or culled over, depending on the degree of infestation.

4. IN COMMERCIAL VEGETABLE PRODUCTION. The light, rich, well-watered soil of southern river bottoms, where important commercial vegetable production is carried on, is highly favorable for root knot development and spread. Almost without exception, anywhere in the South, root knot will soon become a limiting factor to production in such land if it is continuously cropped with susceptible vegetables. Control of root knot becomes essential under these conditions. This can be accomplished by continuously practicing a well-planned rotation in which a root knot-susceptible crop never follows a susceptible one without one or two years of resistant crops between. During the rotation the land should be kept free of weeds since many common weeds are hosts of the root knot worm. In growing sweet potatoes and other crops which are first bedded, the bed soil or sand should be free of the root knot parasites, and in many cases it is worthwhile to make sure of this by sterilizing the bed soil with heat or chemicals as described below. Infested sweet potato mother roots may be disinfested with heat at temperatures that destroy the nematodes without injuring the roots. Some of the other practices suggested for nematode control in field crops are useful in vegetable production, such as summer plowing or fallowing, flooding, and use of green manure and trap crops. Crotalaria is a preferred green manure crop for this purpose. The cost of chemical soil fumigation has now dropped to a point where it may be recommended for vegetable soils of high acre value. Certain foreign tomato species have resistance to the nematodes and are being used in development of resistant commercial tomatoes.

5. IN THE HOME VEGETABLE GARDEN. Where a small plot on the farm is set aside for vegetable production for home use, root knot frequently develops and may seriously lower the vegetable yield. One good preventive measure under these conditions is to fence three adjacent plots with chicken wire and use them in a three-year rotation consisting of vegetable garden, grain, and chicken yard. The chicken house may be set at one end with doors opening to any of the three plots. There should be no drainage from one plot to another. Within a single year a garden spot may be made safe for susceptible vegetables by growing a summer crop of crotalaria followed by a winter green manure crop of oats. Heavy applications of

organic material are helpful. Every effort should be made to prevent the original infestation of gardens by nematodes in purchased tomato, cabbage, or sweet potato plants. Certified plants give this protection. As soil fumigants become cheaper, there is a growing possibility of their use in disinfesting vegetable gardens, as discussed below.

6. IN ORNAMENTAL PLANTINGS. Here above all an ounce of prevention is worth a pound of cure. The soil for such plantings should be free of nematodes or should be disinfested before beginning to plant, and all new bedding stock should be carefully examined for root knot before setting out. Otherwise the disease may not only ruin the value of annual plants, but also spread to prized shrubs, roses, and other perennials. Once these are infested there is very little that can be done to rid them of the disease.

Where root knot is, or is likely to be, a trouble, the owner will do well to specialize on the more resistant ornamentals, such as African marigold, amaryllis, azalea, bittersweet, camellia, day lily, dogwood, dusty miller, euonymus, evening primrose, ferns, gaillardia, holly, lantana, lupine, narcissus, rhododendron, tulip, and zinnia. In some generally susceptible ornamental species, such as dahlias and roses, there are resistant varieties, and susceptible roses can be grown in infested soil if grafted on resistant rootstocks.

In ornamental plantings soil disinfestation often may be justified since the areas are small and their value high. Heat in some form is one of the best means of freeing soil from the root knot worms. If steam is available it may be released under a large inverted pan built for the purpose (Fig. 213), and a half-hour of such steaming is the best method of destroying the worms. Boiling water is a fairly good soil disinfestant if applied liberally on loosened soil.

Where it is impractical to use heat, certain chemicals may be used for destruction of the worms. All of the following soil fumigants have been found to be effective in nematode control: carbon disulfide, chloropicrin (tear gas, Larvacide), DD mixture, dichlorisopropyl ether, methyl bromide alone or in mixtures (Dowfume, Iscobrome), ethylene bromide, ethylene dichloride, pentachlorethane, tetrachlorethane, and xylene. These differ considerably in cost and in ease and safety of application, injury to nearby vegetation, rapidity of evaporation from the soil, and effectiveness against weed seed, weeds, fungi, and insects in the soil. (See p. 464.)

The chemicals are applied in small doses at a depth of 5 to 9 in., at intervals of 1 ft. or more, and the gases formed are confined by watering the surface of the soil or covering it with gas-tight paper. When the cost of the applicator, about \$20.00, makes it uneconomical for the owner of a

small infested plot to purchase one, the applicator may be rented or the work done by a landscape, tree, or termite specialist. Some soil fumigants, such as Garden Dowfume, may be applied simply by dripping the chemical into furrows. The chemicals cannot be used near trees or other permanent vegetation without injuring the plants, and the treated soil must be allowed to air out thoroughly before planting.

7. IN THE GREENHOUSE AND SEEDBEDS. Root knot is inevitably found in greenhouses where scrupulous attention has not been given to sanitation, and even under good greenhouse management it will be introduced occasionally. Often it becomes ruinous to gardenias, snapdragons, chrysanthemums, petunias, roses, and many other ornamentals and greenhouse vegetables. Under greenhouse conditions it is not difficult to control by following a rigorous practice of careful examination of introduced stock, and routine sterilization of soil, benches and pots with steam or an oven (Figs. 212, 213, 214). Where valuable pot plants or bulbs are infected they can sometimes be saved by immersing the pots or bulbs in hot water at 118°F. for $\frac{1}{2}$ hour, but this cannot be recommended as a general practice until more data have been obtained on the heat resistance of greenhouse plants. One ingenious greenhouse operator has reported success in root knot control by closing the empty greenhouse on a hot summer day, and turning on steam, which raises the temperature of the entire house to a point that destroys the nematodes in the soil and on pots, flats, benches, and tools. Soil fumigation by any of the chemicals suggested in the preceding section has a place in greenhouse management. One particularly useful practice is to inject a soil disinfectant about the roots of old greenhouse tomato plants a few days before they are to be removed. This kills the nemas where they are in highest concentrations in and near the roots, and reduces their spread in subsequent working of the soil.

SUGAR BEET NEMATODE (*Heterodera schachtii*)

The sugar beet nematode constitutes a major problem in many sections, especially Utah, Colorado, Idaho, and California. It induces many more small roots than normal, with many dead rootlets. No beets at all, or only small ones are produced. To the roots are attached flask-shaped females similar to those of root knot (Fig. 178). The only other crops affected in the United States are the wild and cultivated crucifers. In Europe, owing to the presence of other physiologic races of the beet nematode, oats, peas, and potatoes are attacked. For controlling the beet nematode one method is a three- to five-year rotation in infested patches or whole fields, with legumes, potatoes, or grains as popular rotation crops. Chemical fumigation of the soil is proving increasingly

useful. A simple but effective form of applicator drips the chemical from the plowsole, plowing and soil treatment being accomplished in the same operation. Fig. 206 shows the destructiveness of this nematode.

WHEAT AND RYE NEMATODE (*Anguina tritici*)

This nematode produces no root swelling but infests stems and leaves making them rolled, twisted, and contorted (Fig. 179). The worms pass



FIG. 178. Nematode infested sugar beet. The small white bodies on the roots are female nematodes. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

up to the growing point, keeping pace with the developing plant, enter the young flowers, mature, copulate, lay the eggs in the ovaries of the grain, and the adults die. The infested ovary develops into a round, hard, dark gall filled with larvae. When the galls are crushed in water the larvae are released and easily seen. In stored galls the nematodes survive nine years or more, but in nature alternate wet and dry periods, in the absence of a susceptible host, soon destroy them. The parasite moves short distances from plant to plant but is introduced into new fields by infested seed. Reinfestation of the new crop is from galls that are planted with the seed, or from nematodes that have lived between crops in the soil. Wheat and rye are the only crops attacked, although other nematodes of less importance attack other grains and grasses. Losses are confined to the more eastern states, and the loss is small for the country as a whole, although on individual farms it may reach 70 per cent of the crop.

Control. Control consists chiefly in the use of gall-free seed in non-infested soil. Infested seed can be cleaned by immersing in a salt solution (40 lbs. in 25 gal. of water), which causes the galls to float, while the good seed sinks. The hot water treatment used for controlling loose smut in wheat (129°F. for 10 minutes) kills the nematodes in the galls. If soil is infested the land should be used for oats, barley, corn, or other crops for one year before returning to wheat or rye. Wheat varieties vary in sus-



FIG. 179. Wheat seedlings attacked by nematodes. Note the wrinkled leaves with curled edges and the emerging leaves tightly rolled. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

ceptibility to the nematode, some, such as Kanred, being resistant. In China, where the nematode is very destructive and abundant in seed wheat, the most practical method of control has been to clean the seed with a nematode-eliminator, an inclined revolving cylinder with a pitted inner wall. The pits are too small to pick up sound grain, which flows down and out while the galls, falling into the pits, are carried up and dropped into an ejector.

STEM AND BULB NEMATODE (*Ditylenchus dipsaci*)

This nematode infests over 300 species of plants. It severely attacks strawberry, red clover, and alfalfa but is best known as a pest of ornamentals, including bulbs in which the trouble is called "ring disease." There are numerous strains of the nematode; some attack a single host species while others have a wider range. The nematode attacks oats, causing the disease "tulip root," and onions, where the disease is called "nematrot" or "bloat." The organism is a soil dweller and survives between crops in crop refuse, bulbs, corms, and tubers and, occasionally, in seed (as

in onion). The symptoms include discoloration or decay of underground parts, and thickening, dwarfing, and distortion of stems, with yields severely curtailed and plants ruined as ornamentals. Spread in the field is chiefly by surface water.

Various control measures apply to the different crops affected. In ornamental plants, hot water treatment to disinfest bulbs or corms, at 110°F. for $\frac{3}{4}$ to 2 $\frac{1}{2}$ hours, is a standard practice and is required as a condition for entry into the United States of some types of ornamental bulbs. Soil fumigation with chemicals is effective where the value of the land and crop justifies the expense. Seed-borne infestation in onions can be controlled by fumigation of the seed with methyl bromide. In red clover, rotation with nonsuscepts is advised, and in alfalfa some progress has been made in developing resistant varieties.

GOLDEN NEMATODE OF POTATO (*Heterodera rostochiensis*)

The potato in the United States is attacked by four species of nematodes. Of these, the golden nematode, while limited in distribution, is most destructive where it occurs, and offers a serious threat to the potato industry if it invades the main potato areas. The yields of the most susceptible potato varieties, such as Green Mountain and Houma, are reduced 70 per cent by attack of the golden nematode. There are no root swellings or tuber lesions, but stunted vines with little productivity. The nematodes remain alive in infested soil for as much as 10 years.

Thanks to early detection and New York regulatory laws, the nematode is still confined to Long Island, but there its gradual spread is arousing the concern of growers. Every effort is being made to check this spread, the recommended measures being to avoid use of tubers from infested fields for seed, field sanitation, soil disinfestation with chemicals, tillage in such a way as to avoid spread by soil or surface water, and barrier strips of nonsusceptible crops surrounding infested fields.

ROOT LESION NEMATODE (*Pratylenchus pratensis*)

In the past *Pratylenchus pratensis* has been referred to as the "meadow nematode." The common name "root lesion nematode," proposed by Godfrey, is preferred, as the nematode is more cosmopolitan than the older term implies. *P. pratensis*, like the root knot and stem and bulb nematodes, affects at least 100 species including such varied economic hosts as strawberry, narcissus, coffee, cotton, alfalfa, tobacco, pineapples, beets, various small grains, corn, turnips, cabbage, and numerous legumes. It is considered one of the most serious of the plant-parasitic nematodes, although it may often be overlooked because of the deficiency type of

symptoms and because no obvious malformation of the roots is apparent. Specific data on losses from this pest are not numerous but the reports include a case in Virginia in which 1300 bushels of Irish potatoes were affected, report of average crop reduction of 28 per cent in tobacco, and accounts of serious losses in coffee, strawberries, chrysanthemums, cotton, and fruit trees. Steiner, a leading authority on nematodes, considers that the overall damage due to root lesion nematodes may be greater than that caused by the root knot nematode.

The nematode attacks the feeding roots with the result that above-ground parts are deprived of water and exhibit symptoms of chronic water deficiency, wilting, yellowing, poor yield, sometimes culminating in death. The appearance of the lesions varies with the host plant attacked but they are generally well-delimited necrotic spots that involve greater and greater masses of the root tissues (Fig. 180). The male and female nematodes are wormlike and similar, about 0.5 mm. in length. The larvae attack the root cortex, forcing their way inward and through the peripheral tissues and destroying the cells along the way. Within the necrotic tissues may be found adults, larvae, and eggs. Frequently the nematode lesions become invaded by secondary fungi or bacteria, which increases the damage and sometimes con-



FIG. 180. Lesions on pine-apple roots produced by the root lesion nematode. (Courtesy, G. H. Godfrey, Texas Agr. Exp. Sta.)

fuses the etiologic conclusions. There is some indication that the root lesion nematode paves the way for the entry of the black root rot fungus into tobacco roots. In the case of affected cereal plants, the roots may be invaded without discoloration and the principal injury may be due to secondary organisms that follow infection by the root lesion nematode. In cotton, susceptibility to wilt in normally wilt-resistant varieties has been ascribed to the influence of infection by the root lesion nematode. Many features of the pathology of this pest are still poorly understood although there is no question of its importance.

Similarly, little specific information is available on control of the root lesion nematode. In coffee the eradication of diseased trees and replanting

with some other crop is prescribed. Infested coffee nursery stock can be disinfested by a hot water treatment (120°F. for 10 minutes). The only recommendation for cereals is rotation with other crops, and manuring. In chrysanthemums, where failures occur due to heavy infestation with this nematode, highly effective control has been obtained by sterilizing the bedding soil with chloropicrin.

Nematodes: Some General Considerations

Physiologic Specialization. In nematodes, as in rusts, powdery mildews, and several other groups of plant pathogens, a parasitic species may consist of several races or strains that appear to be identical. Each race will attack certain plants within the host range of the species and will avoid others. The races sometimes are highly specific. Thus a strain of the stem and bulb nematode from oats attacks only oats and orchard grass but no other of the 100 hosts of this nematode species. A strain from hyacinth would not attack narcissus, and that from narcissus would not attack hyacinth. Other strains are less particular; one from potato, for example, attacks 53 species of plants.

Biological Control. "Mononchs" (species of the nematode genus *Mononchus*) feed on other nematodes. This is probably of little importance as a practical control of parasitic nematodes, since the Mononchs are not restricted to a diet of nematodes, but feed also on rotifers and other soil organisms. They, themselves, are subject to a number of diseases caused by sporozoa and other parasites which frequently destroy the Mononch colonies. *Mononchus papillatus* vigorously attacks even large individuals of the sugar beet nematode, sucking out the body contents. From half-eaten nematodes and other observations, Steiner and Heinly have concluded that *M. papillatus* kills the beet nematode mainly for pleasure, after its appetite is satisfied with other food.

In another predatory nematode, *Dorylaimus*, one species has a hollow spear. It contacts its prey by accident, but at once orients its head at right angles with the surface of its prey, makes a firm contact with its lips, and then suddenly darts in its spear. If unsuccessful, it repeats the process. Once it is in, it is held in place by the heavy muscles of the esophagus, with rhythmic sucking, until the body wall collapses. Nematodes of another genus, *Aphelenchoides*, have still a different system. A small *Aphelenchoides* will approach a large nematode, perhaps twice its length, and quickly slip in and dart its mouth parts into its prey. It may remain attached or may be thrown off by the writhing of its prey, but in a moment the large victim stretches out, paralyzed for several hours by a quick-acting narcotic drug in the saliva of the attacker, whereupon the latter feeds at its leisure.

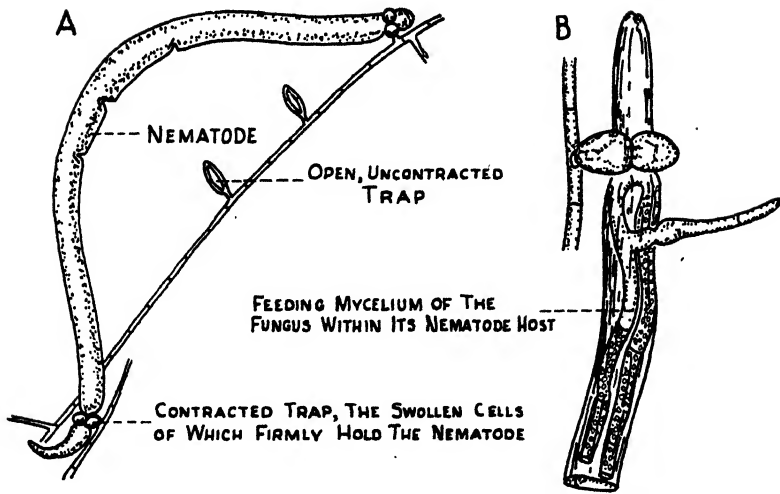


FIG. 181. Parasitism of nematodes by the fungus *Dactylella bembicoides*. (A) A large nema trapped at both ends; its body was filled with fungus hyphae. (B) Details of fungus trap, invading hyphae and an early stage of spore formation of the hyperparasitic fungus. (After Couch.)

Some fungi trap and kill nematodes by the aid of lassolike contraction loops with a trigger action (Fig. 181). Practical use is made of this in Hawaii where the root knot nematode is a serious pest of pineapple. In three to four years of culture, much pineapple waste accumulates. This is plowed under at the rate of 100 to 150 tons per acre. The organic matter stimulates the development of the nematode-trapping fungus, and in a typical case the nematode population dropped from 611 to 2 nematodes per 2500 g. of soil, following the treatment.

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Chapter 14

Physiogenic Diseases

In order that a plant may develop normally, it must not only be free from contagions of infectious disease and depredations of insects and other animals, but it must also be in a suitable environment. The soil must contain adequate but not excessive supplies of water and mineral salts; it must be free of poisonous or toxic substances; the air about the plant must supply an adequate amount of oxygen and be free of toxic gases; the temperatures of air and soil must be favorable to growth and at no time reach excessively high or low levels; and the amount of light available must be adequate for normal growth and yet not excessive. For each plant at any time there is an *optimal* level of each of these environmental variables at which level normal growth occurs. Departure from this optimum in either direction reduces development increasingly as the variable approaches the lowest level (*minimum*) or the highest level (*maximum*) at which the plant can survive. When a plant is in an environment in which one or more of these variables deviates significantly from the optimum, its health is impaired and it is said to be suffering from *nonparasitic*, *abiotic*, or *physiogenic* disease. A great many such diseases affect plants; rarely if ever is a plant in nature growing under fully optimal conditions. While physiogenic causes account for 70 per cent or more of all plant troubles, the amount of discussion devoted to these diseases in this book is very limited in proportion to their importance, since many of them are included in the subject matter of books and courses in plant physiology, field and horticultural crops, and soils.

Diseases Due to Unfavorable Soil Conditions

LACK OF NECESSARY CHEMICAL SUBSTANCES

1. Nitrogen Shortage. The outstanding symptoms of nitrogen shortage are paleness or yellowing of leaves and stems, firing or burning of the lower leaves, stunted growth, and reduced yield with small fruit or seeds. The roots are stunted but less so than the tops. Red pigments often are abundant along veins and stems.

YELLOW BERRY OF WHEAT. The only symptoms are in the harvested grain. Instead of being translucent, the kernels have light yellowish spots

affecting part or all of the grain (Fig. 182). The grain is high in starch but is low in test weight, specific gravity, and protein content, and may be penalized in price. Yellow berry is a very common degrading factor in



FIG. 182. Yellow berry of wheat. (*Left*) Affected grains photographed by transmitted light, showing opaque areas due to starchy contents instead of translucent, protein-rich contents as in the normal grains at the right.

wheat. The etiology of the disease is not well worked out, but the most important factor appears to be the N/K ratio in the soil. Yellow berry in-

creases as soil potash and phosphorus increase, and is eliminated by applications of nitrates. It is best overcome by cropping and cultural practices that conserve and increase the soil nitrogen, such as use of legumes in rotations, suitable green manure crops, summer fallowing, and applications of nitrogenous fertilizers.

2. Phosphorus Shortage. The foliage is dark green in contrast to the yellowing due to nitrogen shortage. Red pigments in those plants that can produce them are often abnormally developed (Fig. 183). The plants are stunted, with short internodes. There is proportionately less stunting of the roots. Irregularly distributed brown patches may occur on the leaves, but the lower leaves usually are not fired as in the case of nitrogen deficiency. Growth is slow, with delayed maturity and small, light fruits and seeds. In root crops, such as turnips and rutabagas, the roots may be dwarfed.



FIG. 183. Phosphorus deficiency in tomato. (1) Normal plant. (2, 3) Slight deficiency. (4-6) Severe deficiency. The dark color of the severely affected leaves is due to purple pigment on the undersides of the leaves, characteristic of this deficiency in tomato. All leaves are of the same age, grown under similar conditions, illustrating the dwarfing effect of phosphorus deficiency. (Courtesy, Dr. H. F. Murphy, Oklahoma A. & M. College.)

3. Potassium Shortage. As with N and P shortages, the plants are stunted, but in this case a chlorotic or bronze and then necrotic spotting or scorching of the leaf tips and margins, advancing up the plant, is characteristic (Fig. 184). The leaves are often crimped or rolled, and the plant as a whole has a rusty or dull appearance. The slender stems may be streaked with brown. Seed often fails to mature or is small. Dieback of the twig tips occurs in woody plants.

COTTON "RUST." Cotton rust is a common and serious trouble due to lack of potassium. The leaves first show a yellowish-white mottling, then yellow spots appear between the veins; these die at the center, brown specks appear around the leaf margin, this dries and curls, and finally the entire leaf is browned and drops prematurely. Many bolls fail to open, and the seed cotton is hard to pick and is of poor quality. Potash-deficient cotton plants are particularly susceptible to *Fusarium* wilt. Control consists in adding enough potash-containing fertilizer to eliminate the symptoms. A common recommendation is 4 per cent potash in a complete fertilizer, such as a 4-12-4 mixture used at a rate up to 200 pounds per acre.

4. Shortage of Minor Elements. Recent studies have shown that plants also require minute quantities of zinc, boron, manganese, and copper for normal development. The amounts required are very small, of the



FIG. 184. Potassium deficiency in corn. Note the chlorotic streaks on the leaves and the scorched leaf tip and margins. (Photograph, American Potash Institute.)



FIG. 185. Rosette, a common and destructive physiogenic disease of pecan trees, easily controllable by applying small amounts of zinc salts. The tufted appearance and numerous dead branches are characteristic.

order of 10 pounds per acre. Lack of these elements produces characteristic and severe symptoms.

ZINC. Pecan trees often suffer from "rosette," a zinc-deficiency disease in which the leaflets are narrow, crinkled, pale, then with dead or perforated areas giving the tree a rusty appearance. The internodes are shortened, giving a bunched or rosetted appearance to the foliage (Fig. 185). The twigs may die back. Affected trees may bear no nuts or only poor ones. "Money-maker" is the only important rosette-resistant variety. Affected trees may be quickly cured by applying zinc sulfate, preferably to the soil. The dosage is $\frac{1}{2}$ to 1 lb. per year of the tree's age or 1 to 2 lbs. per in. of tree diameter, applied around the tree in early spring. Other methods of application include insertion of the dry zinc sulfate in holes bored in the trunk, driving zinc-coated nails into the trees, and spraying the trees in the early spring with 2 to 4 lbs. of zinc sulfate per 100 gal. of water. Several applications are used. The soil applications give the most permanent results. Other zinc-deficiency diseases of a similar type are apple and pear rosette, "mottle leaf" of citrus fruits, and "bronzing" of tung oil trees. Zinc deficiencies are frequently encountered, not only in soils lacking this element but also in soils in which, because of alkalinity or other causes, the zinc present is not available to plants.

BORON. Boron deficiency has been a prominent subject of study in recent years, and many crops have been found to benefit by boron applications. Identification of the trouble depends both on plant symptoms and on chemical or spectroscopic analysis of soils.

ALFALFA YELLOWS. Alfalfa yellows is a widespread boron-deficiency disease. The leaflets are pale to bright or bronzed with green midribs and in severe cases there is cessation of terminal growth. The leaf margins may dry and shrivel. The internodes are shortened and the entire plant stunted (Fig. 186). In the less severe cases the symptoms may show only at the first cutting. Control consists of applications of 5 to 30 lbs. of borax per acre. Larger doses must not be used as they may cause severe boron poisoning of the crop.

Other important boron-deficiency diseases include:

INTERNAL AND EXTERNAL CORK, ROSETTE, AND DIEBACK OF APPLES—precocious ripening; dry fruits, with brown, spongy or corky spots in the flesh; shallow corky patches on surfaces of fruits; rosetting and dieback of the tree.

BROWN HEART OF TURNIPS AND BEETS—brownish, watery or punky core of dead cells in the center of the root as seen in Fig. 187.

DRY ROT OF SUGAR BEETS—dry, spongy decay of the growing heart, ultimately destroying the entire root; adventitious leaves develop around the edge of the heart lesion; large numbers of small leaves with blackened, checked, and twisted petioles are characteristic.

CRACKED STEM OF CELERY—brown mottling of leaves, brown stripes on stems, over which the epidermis breaks and curls back, browning and dieback of roots.

Many other crops show benefit from borax applications, including flax, cauliflower, radish, spinach, potatoes, tomatoes, tobacco, hops, carrots, cotton, asparagus, onions, and cereals.



FIG. 186. Characteristics of alfalfa grown (1) with, and (2) without boron amendment in "yellows" (boron-deficient) soil. (Courtesy, L. G. Willis, N. C. Agr. Exp. Sta.)

MANGANESE. Gray speck of oats is due to manganese deficiency, and is prevalent especially in calcareous soils where manganese is not soluble. "It first appears as light green spots on the leaves, the areas enlarging and changing to buff or light brown color. The spots may be small or large, oblong to linear, depending on conditions and the variety of oats. In some cases the diseased plants are reduced in size." (Dickson.) Applications of manganese sulfate or lowering the soil pH below 7.0 correct the condition. Manganese deficiency also is associated with a disease of sugar cane called Pahala blight, and with chlorosis in various ornamentals.

MAGNESIUM. Deficiency of magnesium produces chlorosis, stunting, and poor growth and fruiting in numerous crop plants. Among the magnesium-deficiency diseases that have received attention are "sand-drown" of tobacco and cotton, a chlorotic condition found in plants grown on badly leached, sandy soils, bronzing of citrus trees, "yellow tip" of pine trees, chlorosis of corn, "white tip" of rice, and mottling of the leaves of other cereals. The most usual means of correcting the deficiency is fertilization with dolomitic (magnesium-containing) limestone and magnesium sulfate.

COPPER. When crops are sprayed with Bordeaux mixture for control of fungus diseases, there is sometimes seen a marked improvement of the crop quite apart from the function of the spray in fungus control. This is now known to be due to the copper. Almost miraculous results have been seen following the use of copper on certain muck and peat soils. A specific



FIG. 187. Brown heart of fodder beets, a serious disease due to boron deficiency. (Courtesy, R. W. G. Dennis, West of Scotland Agr. Coll. Research Bull. 5.)

copper-deficiency disease is dieback or exanthema of citrus trees. Here the leaves are first large, appearing over-vigorous, but soon the twigs begin to die back, or show very small yellowish-green leaves which soon fall. The dying twigs are covered with a reddish-brown gummy discharge. Th:



FIG. 188. Chlorosis in grapefruit, resulting from a combination of zinc and manganese deficiencies, primarily the latter. (Courtesy, A. F. Camp, Fla. Agr. Exp. Sta.)

deficiency is corrected by soil treatment with $\frac{1}{4}$ to 2 lbs. of copper sulfate per acre or by a copper spray.

EXCESS OF SOIL CHEMICALS

A feast may be as bad as a famine in the case of soil chemicals, and excesses of these chemicals may be associated with destructive physiogenic diseases, as seen in the following examples:

1. Excessive Nitrogen. The principal symptoms of excessive nitrogen are overdevelopment of vegetative growth, delayed maturity, dropping of flower buds, sometimes dwarfing, chlorosis, and necrosis. These symptoms do not all appear together, and the appearance of one or more of them is dependent on the state of development of the plant and its

external environment (temperature, water supply) and internal metabolism (protein-carbohydrate relations) at the time excessive nitrogen becomes available. The plants often show increased susceptibility to contagious diseases, such as those caused by rusts, powdery mildews, and bacteria. An example is *bud drop of roses, sweet peas, and tomatoes*, due to excessive nitrogen and increased if at the same time the temperature and moisture are unfavorable. *Niter poisoning of apple*, with marginal necrosis of the leaves is due to excessive soluble nitrates in clean cultivated orchards.

2. Lime-induced Chlorosis. This is characteristic of plants growing in calcareous soils. The plants are sickly and dwarfed, with yellow foliage (Fig. 188). The injury is usually the indirect result of low hydrogen ion content which causes a chlorophyll failure by rendering iron, manganese, boron, and zinc unavailable. The incidence of lime-induced chlorosis depends in part on the developmental stage at which roots make contact with calcareous pockets or layers in the soil. Remedial measures usually consist of addition of iron, manganese, boron, or zinc salts by sprays, injections, or soil treatments.

3. Excessive Acid. Some plants are tolerant of acid soils or even prefer them, but with others, acid soils produce retarded growth, pallor, mottling, or general chlorosis. The roots are poorly developed and often decay. The injury in this case or in that of excessive alkalinity may be due directly to the action of the excessive H^+ or OH^- ions or it may be indirect,



FIG. 189. An alkali spot in a grain field, distinguishable from a root rot spot (Figs. 73, 74) by complete absence of plants, living or dead, within the spot, and increasing degrees of dwarfing as the edge of the spot is approached. (Courtesy, H. J. Harper, Okla. Agr. Exp. Sta.)

due to the effect of these ions on the physical structure of soil or on the solubility of soil nutrients. Control consists in adding the amount of lime necessary for producing an optimum soil reaction for plant growth.

4. Excessive Alkali. Excessive alkalinity results in poor stands, chlorosis, or death (Fig. 189). Crops differ in their alkali-resistance, some such as sugar beets being highly tolerant of alkaline soils. Where the value of the land warrants, the condition may be corrected by applications of gypsum (for "alkalis" consisting of NaCl , Na_2CO_3 , etc.) or sulfur and by cultivation and mulching to retard evaporation and thus keep the alkali from rising to the soil surface.

5. Toxic Trace Elements. The minor elements that are necessary for normal plant development are often highly toxic to plants in doses only a little greater than the amounts required for best growth. *Boron injury* may be apparent when manure is treated with boron-containing larvicides to destroy maggots, and then used on crop land. Other sources of boron are commercial fertilizers, overdoses of borax for correcting boron deficiency, boron-containing irrigation water, and soils that are naturally oversupplied with boron. The principal symptoms of boron injury are retardation or inhibition of germination, stunting or death of plants, leading to poor stands, bleaching or burning of leaves, premature ripening, and lowered yields. Control consists in avoiding the sources of boron listed above, and in leaching the soil. *Copper*, *aluminum*, and *thallium* in excess occasionally prove injurious to plants.

6. Poisoning of Soil by Sprays. In some leading orchard sections where applications of fungicides and insecticides, especially copper and arsenic, have been used abundantly over many years, it is being discovered that the soils around the trees are becoming so impregnated with these chemicals that it is almost impossible to grow cover crops around the trees. The problem is a new one and no satisfactory preventive has been worked out. Leaching by nature in a resting soil or by artificial means may be necessary if such soils are to continue productive. The new organic fungicides and insecticides are a boon to orchardists in that they permit a change in type of chemical deposited on the soil.

WATER DEFICIENCY

Many southern and western crops (cotton, trees, alfalfa) regularly are grown close to the minimal water requirement for survival, and far from the optimum seen, for example, in the 7-ft. cotton of the Mississippi Delta. Their struggle for survival in the water-deficient soils of areas with low annual rainfall is aggravated further by enforced high transpiration rates due to high temperatures and dry air. In the periodic cycles of drought,

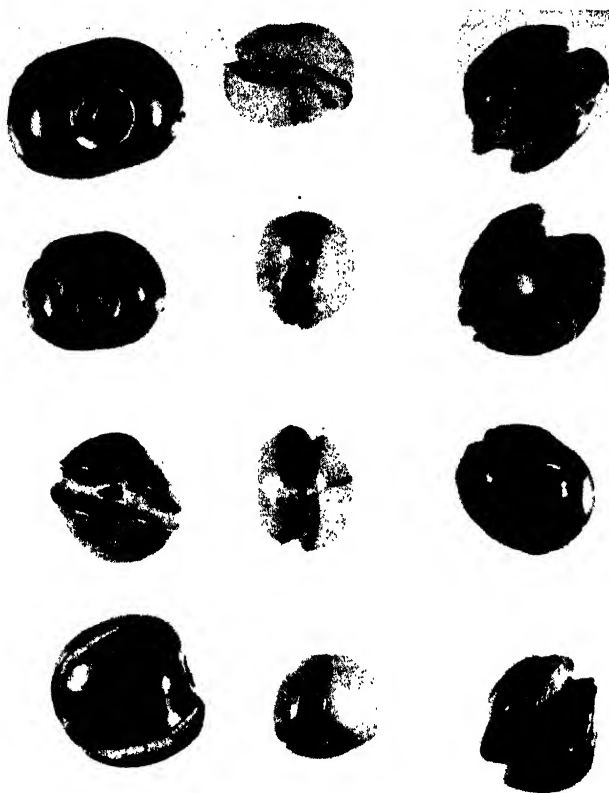


FIG. 190. Cracking in Bing and Lambert cherries at different stages of development; due to rapid water absorption and limited ability of the fruit to expand. (Courtesy, Leif Verner, Idaho Agr. Exp. Sta.)

they display symptoms of *chronic water deficiency*. The leaves are discolored, yellow, red, or brown, and may fall, and the plants are notably stunted. In root crops the roots are undersized, in cereals the grains are shriveled, and in fruit crops the fruits are spotted, deformed, or shriveled. In trees, chronic water deficiency predisposes the trees to attack by borers, which in turn pave the way for wood decay fungi, producing dieback or stag-head, and death. The symptoms often are accompanied by excessive light and heat injury. While this condition is most characteristic of crops grown in the Great Plains area, it may be seen in any area of plant cultivation where occasional droughts occur—indeed, it may be very damaging in moist regions where cultivated species are least adapted to withstand the stress of unusual drought.

In *acute water deficiency* the principal symptom is sudden wilting without stunting or leaf discoloration, as occurs in transplanted plants, plants with sudden and serious root injury, or well-watered plants that are suddenly

deprived of moisture. The various practices which reduce water deficiency include: mulching the soil by shallow cultivation or application of straw or leaf mulches, terracing and other practices that retard runoff, use of drought-resistant crops and varieties such as substitution of sorghum for corn, fallowing to accumulate subsoil moisture, heading back of plants in transplanting, and artificial supplying of water.

EXCESS OF WATER AND IRREGULARITY OF WATER SUPPLY

These factors are responsible for a number of well-characterized plant diseases:

Sunscald of potatoes and many other crops is due to periods of high temperature following an excessive water supply. The tissues, accustomed

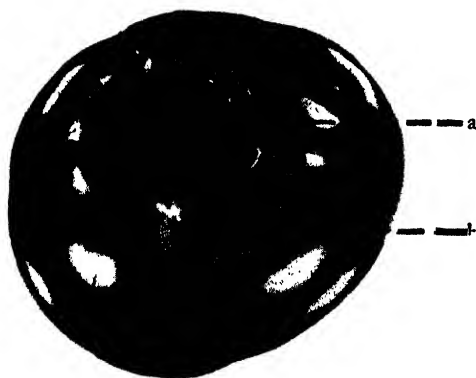


FIG. 191. Blossom-end rot of tomato, a physiogenic disease associated with irregular water supply. Here the rot began as a soft, sunken lesion which later was checked, forming a well-defined corky spot (a); later, when conditions favoring blossom-end rot again occurred, the lesion extended outward as soft decay (b).

to adequate water supply, are suddenly deprived of water without opportunity to adapt themselves to a restricted supply, with resulting blighting or dying of large leaf areas.

Bursting of fruits and hollow heart of potatoes occur when the tissues have been growing slowly during a long dry period and are unable to withstand the high osmotic pressures that develop with the advent of a sudden water excess. Bursting is a common and serious problem in stone fruits (Fig. 190) in areas with continental climates.

Intumescences and edema, as in tomatoes, cotton, and sunflowers, are blisters formed by cells that have burst from sudden water excess after dry periods. The leaves and fruits may be torn and ragged from this cause.

Blossom-end rot of tomatoes takes the form of a sunken, watery, then necrotic lesion at the blossom end of the fruit (Fig. 191). Often this is followed by secondary bacterial or fungus infection, or in mild cases the affected area may cork over without further damage. The fruits ripen prematurely. Blossom-end rot may cause greater loss than any other

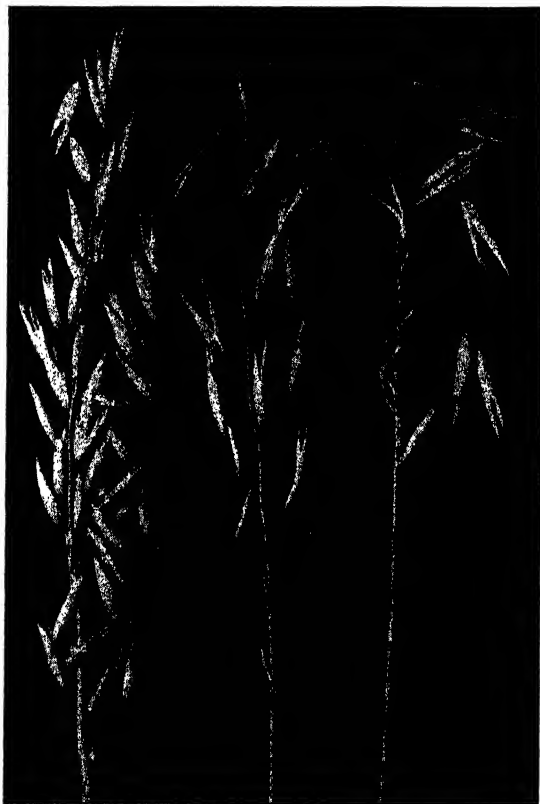


FIG. 192. Blast of oats. On the two heads at the right, many spikelets are blasted; on the head at the left only a few blasted spikelets can be seen. (Courtesy, Illinois Natural History Survey.)

tomato disease in areas subject to extreme variation in rainfall. Its etiology is complex, with the following factors contributing to the disease: a period of drought after the plants have been growing under favorable moisture conditions; lack of water supply due to root killing from waterlogged soil or other causes; shallow root systems; high N, low Ca, low P, and high total salts in the soil; and wind. Control depends on use of the tomato varieties that show some resistance to blossom-end rot, including Marglobe, Pritchard, Marhio, and Michigan State, fertilizing with a good pro-

portion of P in a mixed fertilizer such as 6-10-7 or 6-12-6, addition of humus to the soil, deep soil preparation, frequent cultivation to conserve the water supply, and watering during dry periods.

Alfalfa white spot is a local spotting of the leaf margins or entire blades. It is due to unbalanced water relations and occurs particularly when dry weather is followed by heavy rains.

Blasting of oats and sorghums, or failure to produce seed in an otherwise normal plant, appears to be associated with extremes of water excess or deficiency at blossoming time (Fig. 192). Since in many crops the water supply is uncontrollable, the only recourse is to plant the crops in two or more successive plantings, so that if one planting is affected the others may escape.

Diseases Due to Unfavorable Air Relationships

Plants require adequate oxygen and they are more susceptible to some of the poisonous gases in the air than are animals. Stress at either of these points produces physiogenic diseases.

LACK OF OXYGEN (ASPHYXIATION)

1. Waterlogged Soils. Plants showing typical symptoms of chronic water deficiency often are found in heavily watered locations. The oxygen has been excluded from the waterlogged soil, the roots are unable to respire properly, and as a consequence they lack the ability to take up water even though an abundance is present in the soil. This condition is often found in ornamental plants that have been overwatered, and is the principal cause of dying of crops in low places in a field. The laying of concrete sidewalks may exclude oxygen from tree roots, producing the same effect. The remedy is drainage, lightening the soil with compost or sand, and avoidance of excessive artificial waterings and air exclusion by pavements.

2. Asphyxiation of Fruits and Vegetables in Storage. When fruits and vegetables are stored in deep layers, poorly ventilated, they will often suffer from lack of oxygen or poisoning by carbon dioxide or other volatile products of metabolism. Characteristic and serious storage diseases result. The damage usually increases with increasing temperature, which raises the metabolic rate and increases the oxygen requirement.

BLACKHEART OF POTATOES. Affected tubers often appear normal externally, but on cutting show a black firm core in the center. In advanced stages the black core extends out to the surface, showing as sunken cankers (Fig. 193). In some cases the entire surface of the potato is moist, with a brown discoloration. When the necrosis reaches the surface the

tubers usually show rapid decay due to soft-rot bacteria or other secondary invaders. Blackheart is brought on by the combination of poor aeration and high temperatures. Heavy losses occur in freight cars that are excessively heated to prevent freezing in transit. Preventive measures include storage in shallow layers, not in deep bins, leaving spaces between bins or ranks of sacks, and lowering the temperature of storage to 65°F. or lower



FIG. 193. Potato blackheart. Tuber showing dead tissue breaking through to the surface. (Courtesy, R. S. Kirby, Pa. Agr. Extension Service.)

if possible. It should never be higher than 95°F. Even without special refrigeration equipment, cool temperatures can be obtained by the use of insulated storage houses, preferably sunken in the ground, opened at night and closed during the hot part of the day, with fans for increasing circulation of the air. In the South, potatoes should not be left in the soil after the vines die down, nor exposed to the sun for too long a time after digging.

APPLE SCALD AND INTERNAL BROWNING. These diseases are examples of asphyxiation injuries to fruits. Scald appears as a brown or black discoloration of the skin.

It may be hard or soft, localized in sharply defined areas (as though the fruit had touched a hot stove), or general over the fruit, superficial or deep (Fig. 194). Affected fruits are soon decayed by secondary molds. The trouble is due to the accumulation of metabolic by-products (volatile esters, acetaldehyde, or ethylene) in poorly ventilated storage, rather than to lack of oxygen. It is greatest in immature fruit stored without adequate ventilation and at high humidity and temperature. Green and yellow varieties are more severely attacked than red ones. Control of scald and related troubles depends on allowing fruit to mature and be well colored before picking; storage at once after this stage is reached; protection from sun and heat; storage as near to 32°F. as possible and at 80 to 85 per cent relative humidity; thorough ventilation of the storage compartment; avoidance of tight containers; and wrapping the fruits in oiled paper wrappers, or packing them in shredded oiled paper, or coating the apple skin with wax or oil (Brogdex process). Exposure of apples to a high concentration of CO₂ at the beginning of cold storage has given almost complete control of scald.



FIG. 194. Apple scald, and its control by use of oiled wrappers. Yellow Newtown apples photographed after 9 months of storage. Those at the left were in oiled wrappers; those at the right were not wrapped and show typical scald. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

POISONOUS GASES

Plants may be poisoned by gases in doses so small that they are not injurious to animals and cannot be detected by smell. The chief sources of such gases are factory smoke, illuminating gas, and leakage of gases from refrigerating equipment. The most important toxic gases are sulfur dioxide from smoke, and ethylene from illuminating gas. The reaction of plants may be *acute*, with rapid bleaching of the chlorophyll and death, *chronic*, with a general depression of vital activities, retarded growth, failure to produce blossoms, fruits, or reserve food, early leaf fall, and ultimately death, or *invisible*, with no external symptoms but a reduced development detected only by measuring yield or by chemical analysis. Accurate diagnosis of gas injury is important, as the owners of gas-injured plants are legally entitled to compensation by firms responsible for leakage or liberation of the gas.

1. Sulfur Dioxide (Sulfurous Acid) Injury. Leaves of affected plants show sharply defined brown or red dead areas between the veins, often with a striking color contrast against the green leaf areas (Fig. 195). In coniferous trees the needles become wine red and fall. The injury resembles that from drought, frost, or sun scald, but in these latter cases the dead areas are less sharply outlined. The average person cannot detect less than 3 parts per million of SO_2 by odor, but plants often react to lower concentrations, such as the oak tree (1.4 parts per million). Plant species vary in SO_2 susceptibility. The most susceptible ones include lichens, lupine, clover, bean, pea, alfalfa, and roses. Among the most resistant are beets, potatoes, iris, chicory, and vegetables of the cabbage group.

Grasses and cereals are intermediate in susceptibility (minimal toxic dose about 10 parts per million). Diagnosis of SO_2 depends on: (a) analysis of the SO_2 content of air by chemical methods and comparison with known toxic dosages; (b) analysis of leaves for sulfate or sulfite content; and (c)



FIG. 195. Sulfur dioxide injury to young elm leaf, a result of fumes from an explosives factory one-fourth mile away.

study of the behavior of indicator plants, the best of which include beans, knotweed (*Polygonum*), rhubarb, lupines, and grapes. Prevention of SO_2 injury depends on methods for condensing or retaining smoke and gases in factories, methods for diffusing the gases over wide areas (high stacks, numerous small stacks, etc.), management of smoke discharge to avoid excessive release at any one time and to govern release by wind direction and intensity, and the use of the more resistant plants in smoky areas. Some industries, to avert smoke damage lawsuits, either buy the land about

the factory or sign damage-waiver agreements with nearby property owners.

2. Manufactured Illuminating Gas (Ethylene) Injury. The principal sources are leaky pipes with the primary effect on the plant through gas either in the soil, or in the air. Manufactured illuminating gas also contains carbon monoxide, but the ethylene is 5,000 times as poisonous to plants as the CO. Natural gas, containing methane and ethane but not ethylene, is not toxic to plants. In root poisoning the symptoms are those of general root failure, with water-deficiency symptoms in the leaves plus specific types of curling of the stems and leaves in some plants. Illuminating gas in the air is not a problem outdoors, but often becomes serious in greenhouses and dwellings, especially in winter when ventilation is poor and when the frozen soil permits lateral distribution of the gas for long distances from leaky mains. Carnations, for example, are very sensitive; $\frac{1}{2}$ part per million causes the buds to close, and 1 part per million prevents them from opening. The symptoms are retarded growth, yellowing and leaf fall in dicotyledons, curvatures in monocotyledons, rigor and loss of irritability, curving, even spirals of leaves and petioles. Dormant buds are stimulated to open. (Recall the use of ethylene in forcing potato tubers and woody plants out of dormancy.)

In the past it was customary to use the etiolated sweet pea seedling as the standard test for ethylene, since this plant has a characteristic response to low concentrations of ethylene. Today the standard test plant is the tomato, which is easy to grow and handle, and which responds to 0.1 part per million of ethylene. This is 200 times as sensitive as the human nose, 60 to 100 times as sensitive as the canary (used in mines as a gas detector), and at least 50 times as sensitive as the best chemical test. The response of a 6- to 8-in. tomato plant is a downward bending of the leaves (epinasty). Other useful indicator plants are scarlet sage, mimosa, castor beans, and Jimson weed. Greenhouse operators should keep a few tomato plants near possible gas sources as a routine practice. Control is limited to ventilation, and prompt repairs to leaky gas pipes. In lawsuits to recover property loss from leaky pipes, examination, tests, and testimony by a plant physiologist or plant pathologist usually are necessary.

3. Hydrochloric Acid and Chlorine Injury. HCl and Cl gases from manufacturing processes sometimes are responsible for plant injury. The effect is a sharply outlined killing of leaf margins, in contrast to the interveinal browning due to SO₂. In cases of smoke injury, the effect often is due to a mixture of SO₂, HCl, Cl, HF, and other gases, with correspondingly complicated symptoms.

Diseases Due to Unfavorable Temperatures

HEAT INJURY

The usual symptoms of heat injury are retarded growth, undersized plants, localized burns on leaves and stems, defoliation, premature ripening of fruits, and death. Passing from one part of the country to another, the effect of heat in dwarfing plants becomes noticeable; thus the lilac becomes almost a tree in the North and is often puny in the South, while the reverse is true of privet. Many northern crops, such as cauliflower and broccoli, can hardly be grown in some parts of the South because of a lack of extended cool periods. High temperatures usually are associated with high light intensity and drought, producing disease complexes rather than clear-cut cases of injury due to one of these factors. The direct effect of heat in killing tissues is obvious; less clear is the retarding effect of temperatures only a little above the optimum for growth of a crop. Two factors appear to be involved: (1) "denaturing" of plant proteins which can occur, although slowly, at temperatures as moderate as 95° to 104°F., and (2) alteration of the ratio of photosynthesis:respiration. Supra-optimal temperatures may decrease photosynthesis while at the same time increasing respiration, and if the latter becomes greater than the former, as it will at high temperatures, the plant will lose weight and live only by using up its reserves.

Heat Cankers of Seedlings. In bright sun the temperature at the soil line, where light is absorbed, may reach 150°F. on a summer day. This results in killing of the stem tissues at the soil line, most noticeable on the south sides of stems. It is most important in evergreen tree seedlings and in flax. In the case of tree seedlings it is avoided by sloping the seedlings to the south in transplanting, so that the needles shade the stem during the hottest part of the day, by running the rows north and south rather than east and west, by watering, and by shading with cloth. In flax, the preventive measures include drilling north and south, seeding early so that the plants pass through the seedling stage before the hot weather, and the poor agronomic practices of overseeding or allowing the stand to become weedy, in either case to induce shading of the plants.

Sunstroke. Sunstroke is the outright killing of plants by excessive heat. It is a principal limiting factor in vegetable and flower production in the South. Nasturtiums, for example, regularly die in summer in the South, while in northern states they flourish throughout the summer.

Kelsey Spot of Plums. This is a localized injury of fruits due to temperatures above 100°F. The spots have a sharp margin, depressed center which is often waxy, and purplish red color with dead flesh beneath,

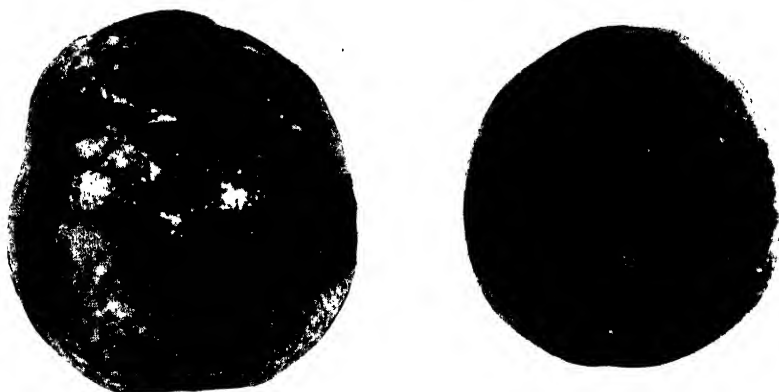


FIG. 196. Kelsey spot of plums (heat injury).

sometimes extending to the pit (Fig. 196). It is distinguished from sunburn in that sunburned areas are diffuse in outline, show no depression, are brown, and occur only in fruits exposed to the sun, while shaded fruits may show Kelsey spot. The only control measure known is to grow a green cover crop which will cool the orchard temperature as much as 4° to 6° in comparison with clean-cultivated orchards.

Tipburn of Potatoes. In this common trouble the leaflets show yellow, then dead tips, the necrosis spreads down the margins and into the blade until the entire leaflet may be destroyed. It is most severe in older leaves and during hot periods accompanied by water shortage. It is distinguished from "hopperburn" caused by the potato leaf hopper, although the two troubles often occur together and are controlled similarly (Fig. 197). Varieties of potatoes differ in their susceptibility to tipburn according to dates of maturity, but the chief measure of control against both tipburn and hopperburn is spraying the vines with Bordeaux mixture. This shades the leaves and thus reduces their temperature, kills young leaf hoppers, and repels the adults. In addition, cultivation and other means of retaining or providing moisture are advised. The Rural types of potatoes have some resistance to tipburn.

COLD INJURY

As the temperature drops from the optimum, growth slows down, no more chlorophyll is formed, red pigments may develop, the tissues are partially dehydrated which gives them greater cold resistance, and with extremes of cold the tissues are frozen and the plant is killed. Freezing injury is too obvious to warrant any prolonged discussion except in the following cases of interest because of their etiology or control.

1. Frost Injury of Fruits. The reproductive parts of blossoms are more sensitive to cold than the remainder of the plant, leading to the

widespread and common sterility of fruit trees which suffer from late spring frost. The control measures used by citrus growers in protecting against frost might be used to a much greater extent by growers of other tree and bush fruits. In many cases the late spring and early fall frosts do not reach more than a few degrees below the safe temperature. Various methods are used for raising the air temperature in the orchard or field this few degrees, such as the use of smudge fires (Fig. 198) or burning logs, especially on the north side of the field, patented orchard heaters, and power fans, or airplanes flying low over the field to stir up the air and prevent it settling in cold pockets. Overhead irrigation or flooding with irrigation water often will save a crop. For frost-susceptible crops, elevated sites with southern exposure are safest from frost injury. In some crops, potash fertilizers are reported to increase cold resistance. The cost of such control measures is not excessive, considering that the entire crop may be saved by such measures, and that in the South particularly the losses from frost are very great because the earliness of the season brings plants out of dormancy before the danger of late spring frost is past. A similar trouble is encountered frequently in wheat, where early-maturing varieties may be sterilized by late frosts. This is one of the reasons for the waning popularity of Early Blackhull wheat in areas growing hard red winter wheat.

2. Winterkilling. Winterkilling due to abnormally low temperatures is especially serious in winter crops or perennials such as small grains, alfalfa, and strawberries. Many factors influence winter injury, such



FIG. 197. Potato hopperburn showing dying and curling of the leaflets. Tipburn is indistinguishable in appearance from hopperburn, and both diseases are controlled by the same means. The holes are caused by flea beetles. (Courtesy, P. E. Tilford, Ohio Agr. Exp. Sta.)



FIG. 198. Protecting an orchard from frost by lard-pail oil heaters. At daybreak the first of two sets of heaters has burned dry and the second set is burning low. (Courtesy, U. S. Weather Bureau, Farmers' Bull. 1588.)

as species and variety of plant, provenience (geographic origin of seed), age (perennials), degree of dormancy or "hardening," kind, time, and degree of pruning, amount of last year's crop, amount of heat and light during the growing season, especially late fall, physical characters of soil and subsoil, fertility and fertilizing practices, soil moisture and drainage, presence or absence of cover crops, and cultural practices such as time and manner of seeding. A control program must consider all of these variables and must be worked out in individual cases by starting with the hardier varieties and working out the other growing practices in ways that will reinforce this hardiness.

The Armistice Day freeze of 1940 which resulted in widespread injury to fruit trees, shrubs, and ornamental trees throughout Arkansas, Missouri, Kansas, and Oklahoma was a striking case of winterkilling. The freeze followed abruptly a period of warm Indian summer and the trees were not dormant at the time when the temperatures suddenly dropped from 85°F. to less than 17°F. within a few hours. The principal injury was to the inner bark of the lower parts of stems and trunks. Affected trees leaved out and blossomed as normal, but as soon as they began to draw heavily on the roots, the injury became evident, exhibited by sudden withering and dying of the foliage followed by death of part or all of the tree. The gumming or bleeding so often seen in branches of stone fruit trees usually is a result either of freeze injury or summer sun scald.

3. Winter Injuries: WINTER BROWNING OF EVERGREENS. When, as often occurs, a cold winter is interrupted by a period of unseasonably warm weather in January or February, evergreens show extensive browning of leaves. This is due to excessive evaporation from the leaves under the influence of warmth at a time when the roots in cold soil are unable to replace the water deficit. It is the common reason for browning of conifers, rhododendrons, and magnolias in early spring and a similar phenomenon accounts for the browning of lawns in midsummer. Deciduous trees show an allied trouble when the warm spell stimulates buds into precocious activity, followed by usual February and March freezes which destroy the unfolding shoots, thus inhibiting flowering and fruiting for that year. An analogous situation is seen in conifers growing in rather cold climates which frequently show a golden yellow appearance on the southern exposure. Evidently one reason for this is that chlorophyll synthesis and chlorophyll photooxidation are differently affected by low temperatures, in this case the pigment being photooxidized more rapidly than new chlorophyll is synthesized, hence the yellowing.

4. Cold Injuries to Harvested Crops. Stored fruits and vegetables are changed, sometimes improved, in composition, color, and flavor by moderate freezing, but are subject to much greater mechanical injury if handled while frozen. Solid freezing is followed by watery disintegration and usually the complete loss of the crop. Potatoes, for example, turn sweet at 32° to 35°F., develop internal frost necrosis (browning) from 28° to 32° and freeze solid below 28°. Control is a matter of providing heat for protection in storage, and it should be noted that public carriers are legally responsible for freezing of crops in transit and must reimburse the shipper for crops destroyed in this way.

Diseases Due to Unfavorable Light Conditions

Plants vary in their light requirements. There are shade and sun plants, but each has its optimal light requirement and suffers from departures from this optimum.

INSUFFICIENT LIGHT

Plants grown in insufficient light are *etiolated*. The internodes and petioles are abnormally elongated, the leaves are reduced in size, the chlorophyll disappears, and there is a suppression of reproductive function. The growth of etiolated plants is succulent, the leaves are thin and wilt easily, the plants are delayed in maturity and show increased susceptibility to certain contagious diseases. Etiolation is sometimes desirable as a horticultural practice, as in the blanching of celery, cauliflower, lettuce,

and cabbage, or in the production of bulb flowers, but in other cases lack of light may be a serious handicap, as in the case of lodging of cereals.

Lodging of Cereals. Lodging or falling down previous to harvest is common in some regions, and rare in others (Fig. 199). There is no single cause of lodging and any of the following factors may be involved: excessive nitrogen, abundance of moisture producing succulent growth, frost injury, attacks of insects or diseases at the crown or foot or along the stems, and mechanical breaking, as by hail. The most important factor of all is lack of light due to overcrowding of the plants which results in weak development of the bases of the culms. Control of lodging depends on lowering the rate of seeding to allow light penetration, cultural methods that avoid nitrogen excess in the soil, and the use of lodging-resistant varieties (Fig. 199). In any locality the adapted varieties of cereals are likely to be more lodging-resistant than unadapted varieties. Much attention is being given to selection for lodging resistance in cereal breeding at the present time, and the results are very encouraging.

EXCESSIVE LIGHT

Although excessive light usually is associated with and complicated by excessive heat and water shortage, there are cases where the light alone is responsible for plant injury, as in the following case.



FIG. 199. Lodging in wheat, primarily a light-deficiency disease, which can be overcome by the use of stiff-strawed, lodging-resistant varieties, as Missouri Early Premium, shown at the left of the photograph. (Photograph, Mo. Agr. Exp. Sta.)

Scalding of bean, cowpea, and soybean leaves. This is common in regions characterized by a high proportion of days with brilliant sunshine, as in Oklahoma, Colorado, and Arizona. On cowpeas the trouble first appears as small red raised spots which enlarge to several millimeters in diameter or coalesce to form extensive blotches, with tan papery centers and a raised, bright red margin. Eventually the entire leaf will die, still retaining some green color, however. The petioles, stems, and sometimes the pods show dead lesions somewhat resembling those from bacterial blight. In beans and soybeans the disease is similar. In beans there is little loss to the seed crop, but in the soybean the disease may seriously lessen the value of the plant for green manure, cover, or forage. No direct control measures are known. The Biloxi soybean is less susceptible than other varieties, and in cases of serious prevalence, the most susceptible legumes should be replaced by those that show less scalding.

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Chapter 15

The Methods of Studying Plant Diseases

When a plant disease problem presents itself two questions are uppermost: What is wrong? What can be done to correct it? The latter depends on the former; the plant pathologist, like the physician, must first make a diagnosis of the cause of disease before he can intelligently recommend control measures.

Field Observations. A study of the disease as it occurs in field, forest, orchard, garden, nursery, or greenhouse will go far toward determining its cause. In making such a study the observer should give attention to many of the following questions:

1. Does the trouble occur on a single crop or is it widespread on vegetation in the vicinity? If the latter, it is most likely an environmental disturbance such as might be caused by drought, gas, or storms.
2. How is the trouble distributed through the field? Is it general over the field, as is usually true of air-borne diseases, or is it in well-defined spots as is often true of soil-borne diseases? If the latter, are the spots at random or are they related to the topography of the land?
3. What loss is the trouble causing? If slight, in comparison with a normal crop, control measures may not be warranted.
4. What are the species and varieties affected and those nearby which are not affected?
5. What is the exact location of the field and the name of the tenant or owner? A map of the distribution of the trouble in the field might be helpful.
6. What are the ecological features of the location: type and fertility of the soil, water supply, slope, exposure, shade, and condition of other vegetation?
7. Are there peculiar features or disturbances in the location, such as digging operations, underlying pipes, excavations, pavement, factory or oil well residues, etc.?
8. What is the history of this year's crop: time and manner of planting, fertilization, watering, pruning, control practices used, previous insect or disease attacks, cultivation?

9. What is the history of the land: has it been rotated; what other crops have been grown here in past years; how did they succeed; has this same crop in this or neighboring locations shown this trouble in previous years and to what extent?

10. What has been the weather record this year and in recent years past: moisture, hail, frosts, temperatures?

11. What was the source of seed or planting stock; where was it grown; was it certified or inspected; are the label or bill of sale and samples of the seed still available?

12. What is the exact nature of the symptoms and signs; are the symptoms systemic, as in cases of root rots, wilt, and many physiogenic diseases, or in the form of local lesions; are leaf lesions at the tips and edges farthest from the veins, as in water deficiencies, or at random over the blade; how does the diseased plant differ from healthy ones in color, size, habit, degree of maturity; are there any signs pointing to the cause of the trouble, such as fungus fruiting bodies, mycelium, rhizomorphs, or sclerotia, molds, mildew, rust, or indications of insect work such as holes, eggs, chewed leaves, droppings, or "sawdust" (frass)?

From this study it may be possible to arrive at once at the cause of the trouble, and if not, the field study often can provide far more information than can be obtained from a laboratory sample.

Plant Disease Surveys. There are many reasons why it is desirable to know the distribution of plant diseases and the amount of damage they are doing in various crops and localities and from one season to another. We must have exact information on the destructiveness of diseases so that funds and energy allotted to plant disease research and extension work may be directed to the most vital problems. We must know the distribution of plant diseases to guide an efficient program of disease control by quarantines and other methods of disease exclusion. We must be informed on the losses suffered by individual growers in order to determine whether the expense of control measures is justified. Up-to-the-minute information on the progress of epiphytotics is useful in directing growers' practices in the immediate future, and reduces financial losses from low prices or speculation due to overoptimistic crop yield predictions. For these and other reasons it is indispensable that pathologists and growers be currently informed on the prevalence, distribution, and destructiveness of plant diseases.

This information is obtained by plant disease surveys. Each state experiment station devotes a small portion of its resources toward securing such data on the leading crops grown. From time to time, as epiphytotics or emergencies dictate, special extensive surveys of a single plant disease

may be made. The need for a national unification of plant disease survey activity became apparent at the time of the first world war and resulted in publication of the *Plant Disease Reporter*, issued by the Division of Mycology and Plant Disease Survey of the U. S. Department of Agriculture. This division guides and integrates surveys on disease problems that are of particular importance. The *Plant Disease Reporter* publishes frequent reports on the progress, distribution, and new outbreaks of plant diseases, annual summaries of the situation relating to disease affecting various crops and losses from plant diseases in leading crops, as well as occasional supplements giving detailed information on epiphytotics and special surveys and studies.

In a few cases the estimates of loss from disease are based on accurate measurements. For example, the dockage records of the federal grain inspectors give a fair picture of the annual loss from bunt in wheat or ergot in rye. But most of the estimates cannot be based on such accurate data but depend on the judgment of well-informed plant pathologists, agronomists, and horticulturists. In most states the final crop loss figures are averages of the estimates of several agricultural workers. In the few cases where crude estimates can be compared with accurate measurements of crop loss, experience has shown that crop observers for the most part have leaned so far on the conservative side that the crop loss figures are much smaller than the losses actually realized. This has been clearly shown in the case of leaf rust of wheat, for example, and the estimates of crop loss by the *Plant Disease Reporter* must be interpreted from this standpoint.

In order for a survey to be efficient it must be extensive, must present a typical cross-section of disease occurrence and, wherever possible, measurements of some sort must be made the basis of estimates. On pp. 23-24 are given the chart and table used in estimating stem rust losses, and aids such as these make for uniformity in the estimates of different observers. Whenever a disease control demonstration is conducted, with comparable plots protected and unprotected, a measurable basis is supplied for future crop loss estimates. Both sulfur dusting of wheat and spraying of potatoes have aided materially in arriving at accurate disease loss estimates in these crops. In the smut diseases, where infected heads represent total loss, sample counts of the percentages of diseased heads give fairly accurate measures of smut losses. The customary practice is to step well out into the field, select a representative spot, and starting at random in the row count 200 to 400 consecutive heads, recording the percentage of those smutted. The same procedure is repeated several times in the field, and the results are averaged.



FIG. 200. Aerial photograph of Texas root rot spots in a cotton field. Airplane surveying and the use of such photographs give fairly accurate measurements of disease damage in cases of those diseases that result in large well-defined spots of dead plants. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

Occasionally, as in certain of the root rot diseases, the spots of diseased or dead plants are apparent in bold relief against adjacent healthy areas, and in such cases airplane surveying is practical and affords a means of fairly exact measurement of disease loss. Fig. 200 is an airplane photograph of a cotton field containing large root rot spots. By using such a photograph and tracing the spots with a planimeter a fairly exact measure of the damage can be obtained.

In other cases it is very difficult even to approach an accurate guess at crop loss because of the varied injurious effects of the disease. In the root, stalk, and ear rot complex of corn, for example, the loss involves many things—stand reduction from seedling blight, devitalization from root decay, reduced food and water transport from stalk and shank infection, lodging, and direct attack on the grain. Actual count of diseased plants may be a poor index of loss because of the various degrees of injury in different plants. Counts of dead plants often are misleading because adjacent healthy plants may compensate partly for the loss in profiting by the additional water, light, and soil nutrients released by the dead plants. Quality as well as quantity of the harvested crop must be considered in the loss estimate. Moreover, the loss estimate is not based on

harvest alone. It includes cost of control practices even where no crop damage is sustained, and it must be corrected for certain savings as in reduced cost of harvesting a diseased or short crop, or compensation for the loss by use of a late-planted substitute crop. When two or more diseases or other types of crop injury affect a crop simultaneously it becomes difficult or impossible to determine the proper proportion of each factor in the total loss. These various considerations show us that in many cases it is very difficult to obtain accurate estimates of crop loss from plant disease. They should not leave us with the feeling that many disease loss estimates are valueless. In spite of these difficulties, the estimates have proved themselves very useful in the past, and as more detailed study is given to individual crops and their diseases the estimates tend to become increasingly accurate. An important phase of pathologic research in the future concerns improved techniques for determining crop damage from plant disease.

Collections. It will often be necessary to make collections for laboratory study. The collected material should include the whole plant or several plants if they are small, and a generous supply of twigs, bark, wood, and leaves if a tree or other large plant is concerned. The plants should be affected but not yet dead. They should be typical of the trouble, not those worst affected unless so indicated. One or more healthy plants should be included for comparison. If these are to be sent to a specialist, they should be wrapped in moist paper and shipped at once. In this case a letter describing the field observations should accompany the specimens. If for laboratory study by the collector, they should be kept moist and placed in a refrigerator until examination.

Preservation of Specimens. If extended microscopic study is intended or a long time must elapse before examination, it may be necessary to preserve the specimens by pressing in a botanical drier or by placing in a preservative solution. Formulas for preservative solutions will be found in the works by Johansen, Rawlins, Riker and Riker, and Sass, cited in the references at the end of the chapter.

Laboratory Examination of Specimens. The first step in identifying diseases in the laboratory is a careful examination of specimens in a search for the less obvious or microscopic symptoms and signs that will permit diagnosis. The low power microscope may reveal such structures as the fruiting bodies of fungi, spore masses, droplets of exuding bacteria, mycelial mats, or the bodies, skins, eggs, or frass of insects. For study with the compound microscope it is usually necessary to prepare a slide mount; in cases where spores are delicately attached and it is necessary to determine the method of spore attachment it may be desirable to use the

low power of the compound microscope and focus directly on the specimen under a strong reflected light. Microscopic mounts may be made in several ways:

1. **SCRAPING** with a scalpel or knife blade to remove spores or epidermis. This material is then transferred to a drop of water on the slide.
2. **TEASING** the tissue apart with two needles in a drop of water on the slide, often best accomplished under the low power microscope. Teasing is especially useful with bacterial diseases, since when the tissues are torn apart the bacterial masses often can be seen streaming out like smoke from a chimney.
3. **SECTIONING** with a hand razor or razor blade to make thin, transparent sections. If soft, the material usually is placed in a slit in a stick of elder pith to hold it during the sectioning. The razor and the pith are flooded with alcohol during sectioning. The sections are floated out in a watch glass of water, and under the low power microscope good sections are transferred with a needle point to a drop of water on the slide.
4. **MACERATION** may be necessary with very hard tissues such as dead leaves or pine needles. The tissue is placed in a little water or potassium hydroxide solution and gently boiled for a few minutes before teasing or sectioning.

Staining is desirable to bring out mycelium and bacteria in plant tissues, and may be indispensable. Various stains are used for the purpose, and these are discussed in references on histologic technique.

Occasionally it may be necessary to germinate spores in order to facilitate the identification of fungi. This is done by placing a spore suspension in a saturated atmosphere. Tap water, rainwater, distilled water, or sugar solutions (1 per cent) may be used to make suspensions. Some of the methods of preparing the germination chambers are shown in Fig. 201.

Identification of Organisms. The first object of laboratory diagnosis of disease is to determine the cause of the disease and, if this is a fungus, bacterium, or nematode, to identify the organism. Space cannot

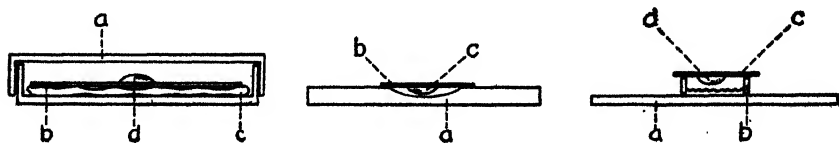


FIG. 201. Three common methods of studying spore germination. (Left) *Petri dish mount*: (a) petri dish; (b) slide; (c) wet filter paper; (d) spore suspension. (Center) *Hollow ground slide*: (a) hollow-ground slide; (b) cover glass; (c) spore suspension. (Right) *Van Tieghem cell*: (a) slide; (b) Van Tieghem cell; (c) cover glass; (d) spore suspension.

be devoted here to a detailed account of classification and identification of these organisms; that is reserved for courses in mycology, bacteriology, and nematology. The following suggestions are given, however, for the convenience of those desirous of pursuing such study.

Fungus pathogens are diagnosed in part by their structure, and in part by their host relations. In groups where the host ranges are narrow, as in rusts and smuts, or where identification of the species is a research problem in itself, as with bacteria, it is pardonable, in diagnosis, to depend largely on the host relations, but final and absolute identification in critical cases depends on a comparison of the organism with all known related species, regardless of host. A number of the most useful reference works are given at the end of this chapter.

Investigating the Causes of Diseases. When a disease is under study and an organism has been found associated with the disease symptoms, it is not always safe to conclude at once that the organism is the cause of the disease. It may be a secondary, saprophytic organism which has gained a foothold in tissues that were diseased from some other cause. At this point it becomes necessary to demonstrate that the organism found is or is not the primary cause of the disease.

KOCH'S POSTULATES. In the case of contagious diseases, proof of the cause of disease follows the principles laid down by the bacteriologist Robert Koch. As applied to plant diseases Koch's rules of proof or postulates are:

1. The organism must be found constantly associated with the observed symptoms.
2. It must be isolated and studied in pure culture, free from all other organisms.
3. Such pure cultures must be used to inoculate healthy susceptible plants and there produce the same disease first observed.
4. From these inoculated plants the organism must be reisolated in pure culture and shown to be the same organism as that present in the original cultures.

It is not always possible to carry through Koch's postulates, since some organisms are obligate parasites and cannot be grown in artificial culture. In these cases (for example, rusts, powdery mildews) the constant association of the organism with the symptoms is taken as proof of the causal relationship, while with virus diseases, infection experiments with juice of diseased plants, insects, or grafting usually are carried out to demonstrate pathogenicity. Koch's rules of proof, while very helpful, do not give infallible evidence of the causation of disease in nature. Sometimes it is possible to demonstrate infection and the recovery of the

inoculated organism under experimental conditions so rarely encountered in nature that for practical purposes the inoculated plant can be considered resistant or immune. In dealing with environmental disease, proof of causation by a given environmental situation is obtained by reproducing that situation under controlled conditions and thereby producing the same symptoms as originally observed.

CULTURE MEDIA: 1. COMPOSITION. Many kinds of nutrient materials are used in culturing pathogens. These are used in either liquid or solid form. Liquid media may be broths, or decoctions of plant and animal substances, or solutions of definite composition. Formulas for these are given by Riker and Riker, cited in the references at the end of the chapter.

For some purposes it is desirable to use sterilized plant tissues as culture media. Stems and cores of potatoes, carrots, and other vegetables are used for this purpose. They are washed, placed in tubes or flasks plugged with cotton, and sterilized.

Solid media are obtained by preparing a broth, decoction, or nutrient solution to each liter of which is added about 20 g. of agar which is a product of seaweeds. It has no nutrient value and has the property of congealing to form a stiff jelly at room temperatures, and of remaining molten at temperatures not high enough to injure most organisms. One of the commonest and best culture media for plant pathogens is potato-dextrose agar prepared as follows:

In 500 cc. of distilled water dissolve 17 g. of agar shreds or powder by very carefully boiling or steaming. Boil 400 g. of diced potatoes in 500 cc. of water and strain. Add the potato decoction to the agar solution, add enough water to make 1000 cc., filter through cloth, and add 20 g. of dextrose.

2. TUBING AND STERILIZING. Although agar may be sterilized and stored in flasks it is customary to pour it into test tubes before sterilizing. The tubes are filled $\frac{1}{3}$ full of the hot agar medium, plugged with a fairly tight roll of cotton, placed in wire baskets and sterilized with steam under pressure or by free steam. In the latter case, the procedure is to heat the medium to 100°C. for 1 hour on each of three successive days. After tubes of agar are removed from the sterilizer they should be slanted at an angle of about 15 degrees and allowed to harden.

ISOLATION OF ORGANISMS IN PURE CULTURE: 1. TISSUE TRANSFERS. In dealing with objects such as fruit or vegetable decays or stem diseases, select a point at the edge of the lesion where it adjoins healthy tissue, surface sterilize the tissue by wiping with a cloth or cotton damp with alcohol (which may or may not be burned off); with a flamed knife or scalpel tear away the outer tissues, exposing the inner lesion, grub out a small bit

of the diseased tissue at the edge of the lesion with a freshly sterilized knife blade, and place on an agar slant or Petri dish. With lesions on small tissues such as leaf spots or rootlet decay, fragments of the lesions may be cut off with a sterile knife, dropped for a minute or two into calcium hypochlorite solution ("B-K" powder, 3 per cent to 10 per cent solution, filtered) and then transferred with a sterile needle to agar.

After a period of from one to several days, fungus or bacterial growth usually will be apparent at the edge of the lesion. If a mixture of organisms is present, places on the plate can be selected where one organism has grown out more rapidly than others and where a fragment of the growth can be picked off with a sterile needle and transferred to a fresh tube or plate.

2. DILUTION PLATES. In dealing with bacteria or fungi that are producing an abundance of spores, pure cultures can be obtained by the dilution plate method. A suspension of spores or bacteria is made with a sterile needle in a drop of sterile water. Three tubes of agar are melted, then allowed to cool until they can be handled freely but before the agar has begun to congeal. A sterile inoculating needle with the end in the form of a small loop is dipped into the suspension and a loopful of spores or bacteria transferred to one of the tubes. It is mixed thoroughly with the agar by stirring with the needle. Then a loopful from this tube is transferred to a second tube and the process is repeated, a loopful from the second tube being transferred to the third tube. The contents of each tube is then poured into a sterile Petri dish and allowed to harden. Later, when the colonies of organisms have grown to such a size as to be readily seen, an individual colony can be transferred with a sterile needle from one of the dishes to a fresh tube of agar, which then will contain a pure culture.

3. STREAKING SPORES OR BACTERIA. A rapid method of securing pure cultures from bacterial or spore masses is to flame a needle, stab it into sterile agar to wet it, touch it to the spore or bacterial mass, then make a series of six to eight streaks across a plate of agar. Toward the end of the streaking most of the bacteria or spores have been rubbed off the needle. and colonies that develop from that area often will be pure.

4. MYCELIUM TRANSFER. If a fungus is producing an abundance of aerial mycelium, individual hyphae may be lifted off with a flamed needle and transferred to agar, and some of these are likely to grow into pure cultures.

5. MONOSPORIC CULTURES. A skilled operator, using a very fine glass needle or pipet under the microscope can pick out individual spores or bacteria and transfer them to agar. Elaborate micro-manipulators can be used to facilitate this work. The spores are smeared first in a thin sus-

pension over a dish or slide of very clear, filtered agar from which they may be selected and transferred.

6. OBSERVING AND STORING CULTURES. Some fungi and bacteria will develop very rapidly on agar; others may require weeks to produce obvious growth. Storage of cultures generally is either at room temperature or in a refrigerator. To maintain cultures alive it is necessary to transfer them to fresh agar within periods of a few weeks to several months, depending on the species. Growth of the organisms will vary greatly with the nature of the medium, the temperature of incubation and other environmental factors.

7. INDUCING SPORULATION. It may be necessary to induce the production of spores in order to identify a fungus in culture. This is done by trying various expedients: changing the temperature, culturing on less nutritious media of different pH levels, suddenly removing the food supply, or matching sexual strains.

INOCULATION OF PLANTS: 1. METHODS OF APPLYING INOCULUM. In some cases attempts are made to duplicate nature's methods of inoculating, but often more or less artificial means are used. Spores or bacteria in suspension may be sprayed on plants with an atomizer, or spores may be dusted on or allowed to fall naturally on moist plants. Mycelium may be placed on uninjured leaves or stems, or the tissues may be pricked, scratched or cut as the inoculum is applied. In grain rust inoculations it is common to rub off the bloom on the leaves with moistened thumb and forefinger, then apply a suspension of spores with a spear-headed needle or spatula. In the case of seedling infection smuts and other surface seed-borne diseases, the spores are dusted on the seed before planting. With soil-borne diseases it is customary to infest the soil. A common method is to grow the pathogen on wheat, meal, or grain and sand, then crumble the medium and mix it with the soil, or simply to prepare a suspension of the organism and water the soil about the plant with this suspension.

2. CONTROL SYSTEM. Each experiment must include plants that are uninoculated but which, in every other respect, are treated as the inoculated plants, even to the extent of laying sterile agar on the control plants, or pricking and scratching them first as with the inoculated ones. Rigid attention must be given to the environment and many failures may be expected where the environmental requirements are neither understood nor met. With many types of inoculation experiments the infection court must be kept moist for from 24 to 48 hours after inoculation as by placing plants in a moist chamber, on a base of wet sphagnum moss, by covering them with plugged lamp chimneys, wet tents, bell jars, or cans, or by binding the infection court with wet cotton or sphagnum. Numerous other

environmental factors such as temperature, light, vigor of the host plant, and fertility of the soil, also play a part in determining the success or failure of infection experiments. Because it is often difficult to maintain all of the requisite conditions for infection, little value can be attached to negative results or failures to produce infection, while positive results must be qualified with the phrase "under the conditions of the experiment," with the realization that under other conditions negative results might be obtained.

3. PRECAUTIONS IN INFECTION TESTS. Apart from attention to the requisites set forth above, an experiment must be managed so as to avoid complicating factors. Insects, diseases other than that under study, mice, rabbits, crows, and slugs should be excluded. In greenhouse tests it is customary to disinfest the soil with steam prior to inoculation unless there is a definite reason for not doing so, as in studying naturally infested soil or competition among the soil organisms. Seed disinfestation may be employed to exclude seed-borne organisms as complicating factors. In some cases the plants are watered with boiled or distilled water to exclude water-borne contagion. Otherwise, the greenhouse or field should be managed in a manner calculated to produce a normal healthy crop.

4. SPECIAL INOCULATION METHODS FOR VIRUS DISEASES. Some virus diseases may be transmitted by rubbing the juice of an infected plant onto the leaves of a healthy one; others can be transmitted only by specific insects or by grafting. Sap transmission is accomplished by rubbing the virus juice gently across leaves, or by stabbing the leaves with needle points wet with virus juice. In refractory cases, the leaves may first be sprinkled with an abrasive such as carborundum. Experimental transmission of tuber and bulb viruses has been effected by cutting out a plug of infected tissue with a cork borer and inserting it in a borer hole in a healthy bulb or tuber. Grafting commonly is used to demonstrate infectivity of those viruses that are not sap-transmissible. In demonstrating transmission by insects it is necessary to rear them in cages, to keep accurate account of the viruliferous condition of each insect or colony and to give proper attention to such points as the length of feeding time required by the insect to acquire the virus and the length of incubation period of the virus in the insect prior to its ability to transmit the virus. Methods of counting viruses, measuring viruses, preparing crystalline virus proteins, and serologic technics with viruses are described in manuals on virus diseases or on phytopathologic techniques, such as that of Riker and Riker cited in the references at the end of this chapter.

Studies on Epiphytology: SEASONAL DEVELOPMENT OF DISEASE. Much may be learned of the epiphytology of a disease by merely observ-

ing it closely throughout the year, noting the prevalence and condition of the pathogen at each season, and correlating this information with the condition of the host and the meteorologic record. Long and painstaking searches may be necessary to determine the condition of the pathogen during dormant seasons, but these searches have been well repaid in the extension of our information on the annual cycles of cereal rusts, bacterial spot of stone fruits, peach leaf curl, and many other diseases. In this connection many diseases are relatively unexplored and a fruitful field of discovery lies ahead of the student who is willing to devote time and careful, persistent observation to this problem.

Searching for a pathogen in the dormant season may be like hunting for a needle in a haystack. To simplify this problem it is customary to gather fallen leaves, fruits, or other plant parts known to be infected during the growing season, enclose them in cheesecloth sacks, tie the sacks to labeled stakes, arrange the tissues in as natural position as possible, and examine them from time to time during the dormant season. A covered chicken wire enclosure aids to protect the material from marauders. In this way it has been possible to discover the perfect stages of many imperfect fungi, to follow the changes that occur during saprogenesis, and to determine the exact details of the resumption of pathogenesis.

DISSEMINATION OF DISEASES. With strictly soil-borne diseases, much may be learned by staking out the outlines of spots of infestation and following the development and spread of the disease during one or several seasons. Important information on the breaking up of Texas root rot spots and its relation to antibiotic action in the soil has been gained in this way. Spread of diseases by water, wind-driven leaves, and insects, can be studied profitably by simple but painstaking means such as will readily suggest themselves. The dissemination of pathogens by wind has received much attention in connection with cereal rusts and downy mildews. A common procedure is to expose vaseline- or agar-covered microscope slides at various altitudes, sometimes from airplanes. From examination of such slides, exposed at critical periods, much useful information has been obtained on the migration of pathogens, the resistance of spores to long periods of exposure in the air, and sources of infection (Fig. 202). Devices to filter and concentrate spores from the air sometimes are used.

RELATION OF DISEASE TO WEATHER. Studies on epiphytology must always be connected with the meteorologic record. The minimal requirement is a continuous record of temperature, air humidity, and rainfall, together with data, if possible, on soil moisture, wind, and light. The records of the U. S. Weather Bureau hold a wealth of useful data that may be correlated with studies on epiphytology, but unless the point of obser-



FIG. 202. Methods of exploring the upper air in studying the migration of fungus spores. (1) F. C. Meier, pioneer in this work (*left*) and E. B. McKinley examining improved Lindberg-Meier "sky hook." (2) Testing equipment used in National Geographic Society flight for sampling the stratosphere for fungus spores. (3) Meier, demonstrating early method of exposing culture dish. (Courtesy, R. J. Haskell.)

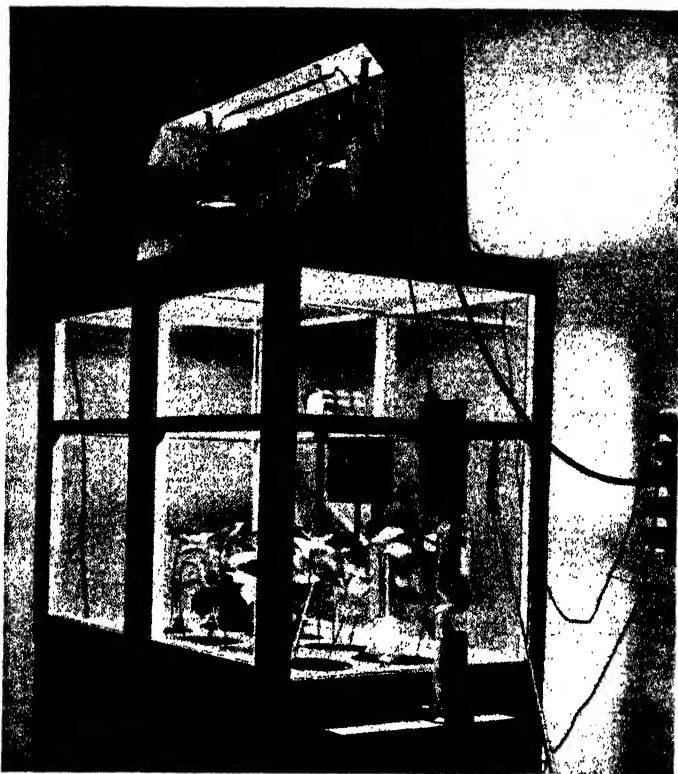


FIG. 203. Equipment for studying plant disease under controlled conditions of temperature, humidity, and light. (Courtesy, Ann. Applied Biol.)

vation is close to a weather station it may be necessary to set up a field weather station equipped with such equipment as thermograph, hygrometer, photometer, and anemometer for obtaining the most pertinent meteorologic information.

PRODUCTION OF DISEASE IN CONTROLLED ENVIRONMENTS. No study of epiphytology is complete without experiments in controlled environments in which each important environmental factor is subjected to controlled variation while the others are held constant. Since the development of the Wisconsin constant soil temperature tanks, numerous types of constant environment equipment have been described, varying from small cabinets to large rooms or greenhouses in which temperature, air moisture, soil moisture, amount and type of light, composition of the soil (often sand cultures with measured nutrient), genetic composition of the host and pathogen, quality and movement of the air, and even other factors are controllable within narrow limits (Fig. 203). Today, knowledge of the role of each of these factors, alone and as modified by others,

is considered essential in a thoroughgoing study of parasitic disease, and this knowledge is being put to good use in interpreting the cause of disease in the field and in providing a basis for control measures.

ARTIFICIAL EPIPHYTOTICS. In determining the varietal resistance or susceptibility of crops toward disease, and in studying the conditions that bring about epiphytotic spread of disease, it is desirable sometimes to induce artificial epiphytotics on a small scale. This is done in various ways. With cereal rusts and mildews in the greenhouse, epiphytotics can be initiated by introducing inoculum and every few days enclosing the bench of plants under a canvas cover overnight after watering the plants and the canvas. In the field the inoculum may be introduced by setting infected plants at frequent intervals, and supplementing moisture with an overhead spray system. Spore production may be stimulated in the inoculum centers by providing them with cloth enclosures at frequent periods. Whether in greenhouse or field, a highly susceptible variety should occur at frequent intervals in the planting.

Studies on Disease Control: LABORATORY TESTS OF FUNGICIDES. Thousands of chemicals that might have fungicidal value can be given preliminary tests in the laboratory when it would be far too costly to test them first in the field. These preliminary tests eliminate the great majority of chemicals from further consideration because of lack of sufficient fungicidal effectiveness or for other reasons. The few that pass the laboratory tests can then be economically tried in the greenhouse and later the field.

Standard laboratory procedures for testing fungicides under strictly controlled conditions have been developed. These are described in the reference cited at the end of the chapter under the title of the American Phytopathological Society. The fungicidal effectiveness is expressed as a fraction or multiple of the fungicidal power of standard Bordeaux mixture. Laboratory assays of fungicides have brought out important features in the behavior of fungicides showing, for example, that when two materials are mixed they may reinforce the fungicidal activity of each other (synergism) or reduce it (antagonism), and that of two fungicides, one may be more active than the other at low dilutions, while the second may surpass the first at higher dilutions.

GREENHOUSE AND FIELD TESTS OF CONTROL PRACTICES. Before embarking on a series of field tests, exposed to the irregularities of weather, field hazards and other vicissitudes, it is customary to undertake greenhouse tests of control practices where plants can be protected and the environment can, to a large extent, be controlled. The greenhouse is adapted for small scale tests of seed treatments, soil treatments, spraying and dusting, and varietal resistance. If the disease is a dangerous one that

might escape to disease-free areas if studied in the field, the greenhouse may be the only place where it can be studied safely. In the field an attempt should be made to secure a site and a planting as uniform as possible, relatively free from one-sided effects or gradients of moisture, soil, shade, wind exposure, or other factors that might make comparisons unreliable between plants in one and another part of the plot. The plot should be as level as possible, free from ditches, or terraces, and should be square or in a broad rectangle rather than in the form of a long narrow strip. In the greenhouse, center benches are better than side benches where accurate comparisons between treatments must be made.

1. CULTURE. The plants should be grown as normally as possible except for the variations involved in the experiment. In the greenhouse, sterilized soil may be used and, unless the test refers to seed treatments, it is well to use suitably treated seed to avoid complications of seedling disease. The plants should be kept free of insects and of diseases other than the one under study. If possible, the plants should be of genetically pure lines, and seed cleaning, grading, or culling of inferior plants is desirable to insure having normal plants in regular stands.

2. INFESTATION. For testing control methods the infestation should be as heavy as it is feasible to make it. Soil in which total losses have occurred in the past, highly susceptible plants as controls, and in many cases artificial epiphytotics or heavy artificial soil or plant inoculations, are useful. To the plant pathologist an epiphytotic or a uniformly heavy infestation of a crop is indeed a "find," since if a control practice proves effective under these conditions its merit is established.

3. STATISTICAL SIGNIFICANCE OF DISEASE CONTROL EXPERIMENTS. Experiments on disease control are nearly always quantitative experiments. We are interested in knowing, not merely that one practice is good and another poor, but just how much better one is than another. We cannot use the expression "this is significantly better than that" as a loose generalization. There is a measuring scale in common acceptance by which we may determine how much weight we may attach to a difference between the results of two treatments.

A brief discussion of statistical significance of experiments is given by Riker and Riker and a more detailed account by Snedecor, cited in the references at the end of the chapter. A properly conducted field test makes use of a plot design that reduces the error from soil or other variations in different parts of the plot and permits one to calculate the odds that the results are significant. With significant results, the odds are at least 19:1 (5 per cent point) that differences in the experimental outcome (yields, etc.) are due to the treatments (spraying, seed treatment, etc.), and not to

chance, while if the odds are 99:1 (1 per cent point), the results are called highly significant.

Literature Review. In any thorough scientific study a student or research worker must be familiar with the literature bearing on the problem at hand, must know of the methods and experimental results of others who have worked on this and similar problems in order to avoid endless repetition of experiments that have already been adequately performed and the embarrassment of announcing a "discovery" that may have been made by some other worker long ago. The literature of plant pathology is vast, scattered through thousands of American and foreign scientific journals, bulletins, circulars, and books. Fortunately, we have a number of very useful guides to this literature so that the problem is not as baffling as it first seems.

Let us suppose that the problem concerns the speckled leaf blotch disease of wheat caused by the fungus *Septoria tritici*, and that it is desired to trace down all the papers dealing with earlier studies on this disease. One would first turn to certain journals that have well-indexed lists of papers dealing with pathology and through their indexes locate and record the titles of the papers concerned. In the indexes one would search for reports of the disease under five key headings:

Wheat: *Septoria tritici*

Wheat: Speckled leaf blotch

Triticum (the scientific name of wheat): *Septoria tritici*

Triticum: Speckled leaf blotch

Septoria tritici.

The most useful indexing journals for this purpose would be the *Experiment Station Record*, *Biological Abstracts*, *Review of Applied Mycology*, and the *Agricultural Index*. The first three give not only titles of papers but also short abstracts of their contents. If the original paper is available in a nearby library, one should not be satisfied with reading only the abstract, although this will be useful in indicating whether the original paper should be consulted. For the journal *Phytopathology*, which contains many important papers, there is a 30-year index covering the period from 1910–1940, which greatly simplifies the search for papers in that journal. *Mycologia*, another major reference journal, has a similar index for 1909–1932. Many departments of plant pathology also maintain card indexes of plant disease literature that will be particularly helpful, for as a rule they are more up-to-date than the indexing journals. Usually, the scientific papers consulted will have limited bibliographies included, from which further titles on the problem may be gleaned. The bibliography is best kept on filing cards,

one card for each title. Each title should present complete information on the source, as in the titles of references at the end of each chapter in this book.

Guided by the bibliography one can now turn to the papers and bulletins themselves. In searching a library for technical papers one will find that these rarely are listed in library card catalogues by author or subject. Instead, one must search for a file of the journal or bulletin concerned, then turn to the proper volume and page. From each article one will want to record data on methods, experimental results, and conclusions, at greater or less length according to one's interest in each phase of the problem. Notes may be recorded on the filing card bearing the title of the article concerned. To help in later organization of the information, symbols indicating the phases of the problem covered in that article may be entered on the card. Later, the entire body of information may be arranged in an organized fashion, one of the most useful of which is the Cornell outline of a plant disease study, given below in slightly modified form:

1. Hosts
 - a. Plants affected
 - b. Varietal susceptibility
2. Disease
 - a. Names
 - b. History and range
 - c. Importance
 - d. Symptoms and signs
 - e. Etiology
 - (1) Name, history, and classification of the pathogen
 - (2) Pathogenicity
 - (3) Life history (including pathogenesis and saprogenesis)
 - f. Epiphytology
3. Control
 - a. By regulation
 - b. By the use of host resistance
 - c. By cultural methods (including chemical and physical treatments)
4. Bibliography

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For Determining What Genera and Species of Pathogens Have Been Found on a Given Host:

- Anderson, P., *et al.*: Check List of Diseases of Economic Plants in the United States, *U. S. Dep. Agr., Bull.* 1366, 1926. (Now being revised by F. Weiss and published in the *Plant Disease Reporter*, 1940-1947.)
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For Diagnosis of Pathogens Affecting Given Classes of Crops (Field Crops, Fruits, etc.):

See references on pp. 19-20.

For Diagnosis of Pathogens Causing Given Types of Disease (Smuts, Rusts, etc.):

See references at the end of the chapter dealing with the given type of disease.

Chapter 16

Environment and Parasitic Disease

For contagious disease to occur in plants it is not enough that a susceptible host and a virulent pathogen come in contact. *No disease can result unless the environment favors its development.* Rust spores are present in every field of susceptible wheat, but only under a favorable set of environmental conditions can an epiphytotic result.

It was not long ago that growers and scientists alike ascribed each plague or crop failure to environment alone—to soil deficiencies, to poor seed or degenerate seed stocks unable to cope with their environment, to errors in cultural practices, or to drought, rain, heat, or cold. All these are vastly important; alone they account for many plant troubles, and as they present the complex of conditions necessary to host and pathogen they set the stage on which the tragedy of parasitic plant disease can be enacted. The record of plant disease is far more than a record of pathogenic organisms with their formidable Latin names; it is a record of the excesses of these pathogens under the influence of environmental combinations that permit and encourage their most devastating spread and destructiveness. The geographic distribution of plant diseases, club root of Crucifers in the North, root knot in the South, Texas root rot in the Southwest, and *Igniarius* heart rot from the Arctic Circle to the tropics, all bear telling witness that plant disease can become rampant only if virulent pathogen and susceptible host find themselves in an environment that will permit the one to flourish at the expense of the other. Even more striking evidence of this is seen in those enphytotic diseases that are always present in trifling amount, but that at long intervals, when the many necessary factors of an exacting environment combine, burst forth in almost unaccountable fury.

The environment may favor the development of disease:

1. *By its effects on the parasite*—moisture to permit spore ejection and germination; wind to distribute inoculum; temperatures at which the parasite flourishes, etc.
2. *By its effects on the vector.* Diseases that depend largely or entirely on insect vectors, as bacterial wilt of corn and curly top of sugar

beets are dependent for their annual occurrence on the geographic distribution of the vector, which in turn is limited in its prevalence and distribution by environmental factors.

3. *By its effects on the host*, favoring either susceptibility to infection or the subsequent development of the disease, as by promoting succulence, extending the period of exposure, devitalizing it, etc.

The effect of environment on disease is a complex one consisting of many factors (temperature, moisture, nutrition, light) interacting with one another and affecting both parasite and host simultaneously. For example, the temperature producing most rapid development of the parasite will not necessarily produce the greatest amount of disease. The environmental background producing most destructive disease is often intermediate between the optimum for parasite development and the optimum for host susceptibility. The optimum level of any single environmental factor in producing disease may vary considerably with the changing level of some other factor.

These principles describing the effect of environment on parasitic disease are brought out in the following examples:

Effect of Temperature on Parasitic Disease: 1. GIBBERELLA SEEDLING BLIGHT OF CEREALS (THE SEEDLING-BLIGHT STAGE OF CEREAL SCAB). Studies were made by Dickson in Wisconsin of the effect of soil temperature on seedling blight. Wheat and corn plants were grown in watertight pots immersed in tanks of water, with thermostatically controlled temperature. The effect of temperature on the fungus alone in pure culture was observed in constant temperature incubators. The results are indicated in Fig. 204.

OBSERVATIONS:

- a. The fungus grows best at moderately high temperatures.

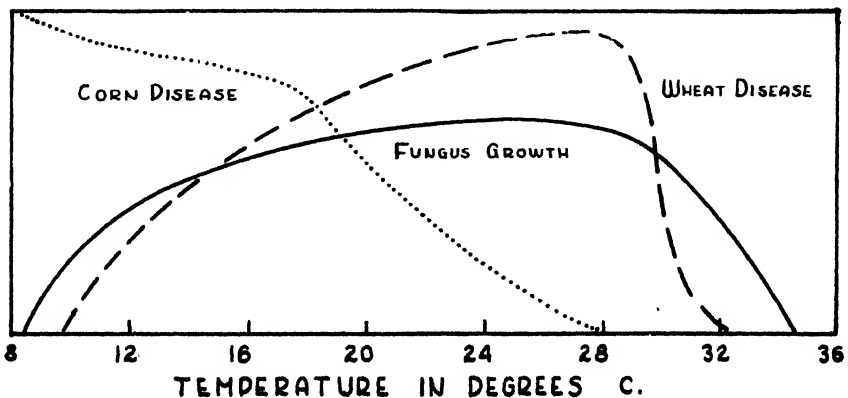


FIG. 204. Relation between soil temperature and development of seedling blight in wheat and corn. (After Dickson.)

b. The most severe blight in *corn* occurs at low temperatures which are least favorable for either the growth of the fungus or the usual growth of corn.

c. The most severe blight in *wheat* occurs at higher temperatures which favor the fungus but are least favorable for the normal growth of wheat.

CONCLUSIONS:

a. The greatest amount of disease is not necessarily at the temperature which is most favorable to the fungus (cf. corn).

b. In this case the effect of temperature is primarily on the host plant, rather than on the fungus, unfavorable temperatures for normal plant growth predisposing the plants to disease.

c. The disease may be controlled in either crop by planting at a date when the soil temperature is best suited for the normal growth of the crop, i.e., planting wheat in cool soil, planting corn in warm soil.

2. BUNT (COVERED OR STINKING SMUT) OF WHEAT: OBSERVATION:

For the disease to occur, the bunt spores on the seed surface must germinate at the time that the seedling is sprouting. Bunt spores germinate best in the range 45° to 55°F., fairly well up to 61°, and poorly or not at all above 65°F. Wheat develops best at 61° but will germinate at soil temperatures as high as 81°F.

CONCLUSIONS:

In this case the effect of temperature is primarily on the parasite. Bunt will be most serious when infested seed germinates in cool soil favorable to the best wheat development. Bunt can be entirely avoided by planting when the soil is quite warm (65°F. or higher). This explains why early-planted winter wheat or late-planted spring wheat often escape the disease, but planting in warm soil is not recommended for bunt control since warm soil does not favor the best wheat growth, increases Hessian fly damage, and predisposes the plant to seedling blight and foot rots. Instead, for the best yields, plant the grain in cool soil even though this favors bunt infection, and control the bunt by one of the highly effective and inexpensive seed treatments.

Effect of Moisture on Parasitic Disease. Moisture simultaneously affects both host and parasite and in this joint effect exerts a profound influence on the development of disease. Fungus spores usually require moisture for germination but vary widely in this requirement, from the urediospores of wheat leaf rust which must have a film of water in order to germinate to the conidia of some powdery mildews which will germinate at zero humidity. Some spores are forcibly ejected only in presence of high moisture. Bacterial pathogens require water droplets for spread. Once a pathogen has gained the interior of a plant it becomes relatively independent of external moisture, the internal moisture of plant tissues sufficing

for development of the organism. Abundant moisture favors rank, succulent development of the host which in many cases predisposes the host to infection. At the same time, abundant moisture in dry-land areas may so stimulate plant growth that the damaging effects of disease are obscured. (Example: Rust years are wet years, in which the increased production due to adequate rainfall disguises the losses from rust.) The effects of excess moisture are seen also in the destructiveness of contagious diseases in dense, overplanted stands which result in moist air about the plants, and in culture under irrigation or in wet climates, where rusts, powdery and downy mildews, and other diseases often become the deciding factor in production. Deficient moisture after infection has occurred, as in wilt diseases or stem rust, may greatly aggravate the damage to diseased plants.

Effect of Wind on Parasitic Disease. Wind is of chief importance in spreading inoculum of disease (spores, bacteria in wind-driven rain, wind-blown leaves, insect vectors), and as such is highly effective. (Recall that rust spores can initiate infections and epiphytotics after having been blown for hundreds of miles in the upper air.) Wind is of importance also in influencing disease by its effect on moisture and temperature (accelerating evaporation), and by producing whipping injuries that permit infection by wound parasites.

Effect of Light on Parasitic Disease. Insufficient light often predisposes plants to disease by increasing the succulence or decreasing the vigor of plants. Most parasites are relatively independent of light, fungus spores, for example, usually germinating equally well in light or darkness. The day and night alternations in spore production or mycelium spread seen in brown rot of stone fruits and agar cultures are due usually to diurnal temperature fluctuations rather than to variations in light.

Effect of Soil on Parasitic Disease. Many examples will be recalled of infectious diseases which occur only in certain types of soil, or which may be controlled by changing certain of the soil properties.

1. **FERTILITY.** All contagious plant diseases fall into two groups, those which attack the most vigorous hosts (rusts, powdery mildews, virus diseases) and those which can attack only weakened plants (dry-land foot rot of wheat and numerous other root rot, canker, and leaf spot diseases). The organisms that attack only vigorous plants usually are obligate parasites dependent on living cells for support. Many bacterial diseases flourish in rapidly growing tissues and slacken their development in hosts that are weakened by soil deficiencies of any sort (e.g., fire blight of apple and pear).

2. **N-P-K RATIO.** Excessive nitrogen in general lengthens the vegetative period which may increase the destructiveness of contagious disease by lengthening the period of susceptibility (stem rust). In some cases the

reverse effect results, the longer growing period enabling the plant to make additional growth to compensate for that lost through disease (potato late blight). Phosphorus deficiency lengthens the vegetative period; amendments of phosphorus correct this, and with potassium stimulate the development of strong mechanical tissues in contrast to the succulent tissues resulting from nitrogen fertilization. Thus, by their own action, or in counteracting the effects of nitrogen, phosphorus and potassium in fertilization often afford effective control of those diseases that are favored by high nitrogen. Exceptions to this rule include the bacterial spot of stone fruits, which is controlled practically by applications of readily available nitrogen, rust of flax, a crop which is very dependent on phosphorus for normal growth, and suffers most from rust in soils with a low N/P ratio on this account, and seedling infection smuts in which high nitrogen promotes such rapid growth that the growing point grows away from the smut mycelium and produces healthy heads.

Cotton wilt is an outstanding case of a parasitic disease so related to potassium deficiency in the soil that applications of potash are a major feature of the control program. A similar relationship exists between cabbage yellows and potassium.

3. SOIL REACTION (PH). Like higher plants, plant parasites vary in their pH requirements, and there is no logical classification of plant diseases according to pH requirements of the pathogens. Within the same genus are found acidophilic species (*Fusarium vasinfectum*—cotton wilt) and basophilic species (*Fusarium nivale*—snow mold root rot of wheat). Examples of pH requirements in certain diseases are given in Fig. 205.

There is a long list of contagious diseases favored by acid soil. These include:

COTTON WILT, possibly because potassium is less available in acid soils.

TOMATO WILT, which decreases with rising soil pH.

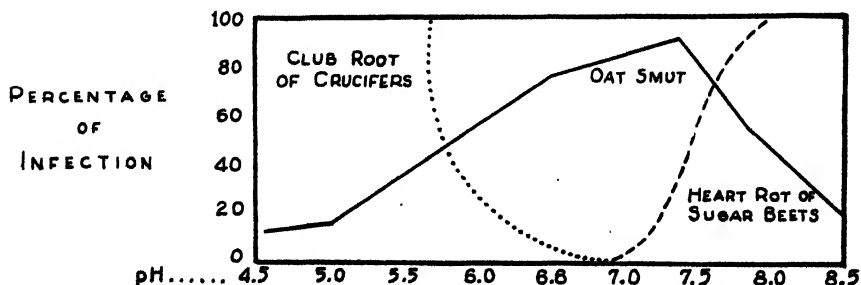


FIG. 205. Relation of the development of three diseases to pH of the soil. (After Gäumann.)

RHIZOCTONIA ROOT ROT, which is controllable by physiologically basic fertilizers.

CLUB ROOT OF CRUCIFERS (see Fig. 205). This is a classic case of a disease which is completely controlled by liming acid soils to raise the soil pH to 7.0 to 7.5.

On the other hand, numerous other contagious diseases are favored by alkaline soil reaction, e.g.:

TEXAS ROOT ROT, which occurs on every soil type in Texas but is most severe on calcareous soils and to some extent can be combated by soil acidification.

POTATO SCAB, a classic case in which the disease increases as the pH value drops from 8.5 to about 5.7 and then falls off sharply, so that a pH of 5.2 to 5.4 is quite unfavorable to the disease. It is practically controlled in some areas where the soils are slightly acid by further soil acidification with 300 to 500 lbs. of sulfur per acre.

BLACK ROOT ROT OF TOBACCO, which is most severe in limed fields.

SOIL ROT OR POX OF SWEET POTATO, which is controlled by adding sulfur to the soil to lower its pH.

Practically, diseases such as these are favored by acid or alkaline soils and are often controllable by altering the soil reaction, but actually little is known of the mechanism of this relationship. Thus, adding lime to soil might affect disease in many ways, such as altering the availability of other elements influencing disease, affecting either host or parasite directly, disinfecting the soil, changing its physical properties, and others.

4. SOIL TEXTURE. Many plant diseases are characterized by restriction to light or heavy soils. In root knot, for example, the lighter soils favor the migration of the pathogens from one plant to another. In other cases heavy soil, through its retention of water or exclusion of oxygen, influences disease either by changing the susceptibility of the host plant or providing a more favorable environment for activities of the pathogen. Bacterial blight of cotton in dry-land areas is most destructive on plants in the heavier soils, since these soils are more retentive of water which favors the succulent type of growth most susceptible to the blight bacteria.

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Chapter 17

Etiology and Epiphytology of Disease

Etiology

In connection with attempts to control a plant disease it is often helpful to study the cause of the disease and, if the disease is due to a pathogen, to work out its habits and cycle of activities. This study of the agencies which cause disease is called *etiology* (Greek: *aitia* = "cause").

Names of Pathogenic Organisms. Just as the common names of flowering plants vary in different regions, so the common names of diseases vary from one locality to another and often confuse one disease with another. Most pathogens have no common names; instead, scientific names, uniform the world over, are used in their designation. An example is *Phymatotrichum omnivorum* (Shear) Duggar, the pathogen causing Texas root rot. *Phymatotrichum* is a genus of sclerotia-forming imperfect fungi, the name coming from two Greek roots, *phyma* = tubercle + *trichos* = hair, evidently referring to the mycelium and sclerotia. The species name, *omnivorum* ("feeds on everything"), refers to the very wide host range of this fungus. When first discovered, this fungus was found in a sterile form, without spores, and was placed in *Ozonium*, a genus of imperfect fungi bearing no spores, by its discoverer, Shear, whose name, according to custom, thereafter followed the scientific name, *Ozonium omnivorum* Shear. But when the spore mats were discovered the pathogen was placed in a spore-forming genus of imperfect fungi, *Phymatotrichum*. Shear's name is retained in parentheses to show that he was the one who originally described the fungus and gave it its present species name, while this is followed by the name of Duggar who discovered the spore mats and gave the fungus the name it now bears, *Phymatotrichum omnivorum* (Shear) Duggar. In elementary textbooks and other nontechnical publications the name of the authority who originally described a pathogen is usually omitted, as has been done in the preceding chapters. But in scientific papers each technical name of a pathogen must include the authority, whose name is ordinarily abbreviated, e.g., *Puccinia graminis* Pers. [Persoon] or *Puccinia trititica* Eriks. [Eriksson].

Large and complex species often are subdivided into structurally or

physiologically distinct varieties which are given names also, such as *Puccinia graminis* variety *tritici* (stem rust of wheat), *P. graminis* variety *avenae* (stem rust of oats), and *P. graminis* variety *hordei* (stem rust of barley). In fungi which show physiologic specialization the species or variety is further subdivided into numbered forms or races, as *Puccinia graminis* variety *tritici*, race 56, or the bean anthracnose organism *Colletotrichum lindemuthianum*, form 3.

History of Pathogenic Organisms. The history of a *disease* (crop injury) and the history of its *pathogen* (fungus, bacterium, etc.) may coincide if the pathogen and disease were discovered at the same time and studied by the same persons. But in many cases the history of the organism and that of the disease are entirely distinct. Thus the history of stem rust, the disease, goes back to epiphytotics in Bible times, while the history of its pathogen, *Puccinia graminis*, begins with the discovery and naming of the fungus by Persoon in 1797. Conversely, the honey mushroom, *Armillaria mellea*, has been known as a fungus at least since 1821 and its history as a fungus dates from that time or earlier, but the history of the root rot disease caused by *A. mellea* only goes back to its pathologic study beginning about 1874. Even at the present time the history of a pathogenic organism, as such, and that of the disease it causes are progressing in different directions. That of the pathogen is concerned with its structure, habits, physiology, and classification, while the history of a disease has to do primarily with the effects on the host and on the crop, its epiphytology, and control.

Life-history of a Pathogenic Organism. This is the story of all the activities through which it passes. Many pathogens spend part of their life cycle as parasites and part as saprophytes. The stage during which the organism is associated with the living tissues of its host plant is called *pathogenesis*, while the period during which it is not living in vital association with the living tissues of the host, and may be either dormant or living as a saprophyte, is termed *saprogenesis*. The sum of all activities during these two periods constitutes the life history of the pathogen.

1. **PATHOGENESIS.** Pathogenesis is the period from the time at which inoculum is first deposited on the host plant until the final reaction of the host, the death of its tissues, and the beginning of a dormant period or saprophytic existence of the pathogen on the dead remains of the host. Pathogenesis consists of several stages:

INOCULATION is the transfer of *inoculum* (spores, fragments of mycelium, seeds, eggs, or any other reproductive body) from its source to an *infection court*, any place where infection can occur on the new host. The transport of inoculum from a diseased plant to a healthy one is *dis-*

semination and is accomplished by an agent of dissemination or *vector*. Dissemination may be either by *continuous spread*, the disease progressively passing from one plant or field to the next, or *discontinuous*, traveling in a long jump from one part of the world to another.

Continuous Spread. This is accomplished by various types of vectors, the leading ones being (1) wind, (2) wind-driven rain, (3) water, (4) insects and other animals, (5) agricultural machines, (6) contact of plants, (7) wind-blown leaves, and (8) man himself.

Wind is by far the most important vector in the case of spore-borne fungus diseases. While wind is the principal agent of continuous spread as in the case of rusts, leaf spots, mildews, smuts, and numerous other types of fungus diseases, it can act also as an agent of discontinuous spread, as has been shown in the trapping of viable fungus spores from airplanes over the North Polar regions, and the Pacific Ocean (Fig. 202).

Wind-driven rain is the most important agent of continuous spread of bacterial diseases, such as bacterial blight of cotton. It has been shown that the droplets from a splashing raindrop will drift as much as 20 ft. in quiet air, and even a light breeze will greatly increase effectiveness of this type of spread.

Water in the form of irrigation, surface runoff, streams, and public water supplies often is instrumental in the spread of the typically soil-borne disease organisms, such as the root rot and damping-off fungi, and nematodes, and also in the case of bacteria, e.g., those which cause alfalfa wilt.

Insects and other animals play a large part in the dissemination of virus, bacterial, and fungus diseases, and sometimes are the only natural means by which a disease may be spread. Examples are the transmission of sugar beet curly top by the sugar beet leaf hopper, fire blight of apple and pear by bees, the Dutch elm disease by bark beetles, and mistletoe by birds.

Agricultural machines are an important agency of spread of diseases by movement of contaminated soil on wheels, or by carrying the organisms of disease as on the blades of a mower. For this reason, diseases often spread most rapidly in the direction of cultivation (root rot diseases, alfalfa wilt, nematode diseases, Fig. 206).

Contact of plants often is necessary in the spread of disease fungi which produce no spores but spread by slow mycelial growth from one plant to the next, as in the case of Texas root rot and *Rhizoctonia* root rot of many plants.

Wind-blown leaves at times may become efficient vectors of continuous spread. Rolfs has shown that cotton leaves carried by a whirlwind can spread bacterial blight effectively over a distance of $\frac{1}{2}$ mile or more.

Man himself can be a very efficient vector in his operations of handling



FIG. 206. Sugar beets on land severely infested with nematodes. This field well illustrates how nematode infestation is spread in the direction of irrigation and cultivation. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

plants. In picking beans when the vines are wet the hands distribute the bacteria of bean blight so effectively that the greater part of the crop may be lost through this practice. Likewise, the handling of tobacco or tomato plants in operations of transplanting, pruning, staking, and hand pollinating often is followed by 100 per cent infestation with tobacco mosaic.

Discontinuous Spread. Discontinuous spread is due largely to the activities of man who is responsible for the transport of disease from one part of the country or world to another by shipments of *infested seed* (smuts, ergot, dodder, wheat nematodes, bean blight and mosaic), or *other propagating parts* (viruses and nematodes in potato tubers or flower bulbs; various diseases of nursery stock and cuttings), of *botanical specimens or experimental material* (as in the introduction of the gypsy moth to America), of *cut flowers and plants*, and of disease-infested *agricultural products*, such as cotton lint and binders, or elm tree burls which brought the Dutch elm disease from Europe to America. In recent years the development of modern transportation, with the extensive use of the automobile and airplane, has favored the discontinuous spread of disease both by carrying disease organisms for long distances on such vehicles, and by increasing the difficulty of preventing the dissemination of infested plant material by inspection and quarantines.

THE INCUBATION PERIOD extends from the moment at which inoculum is deposited in the infection court until the moment of the first observed reaction of the plant to the pathogen. It may be very short, a matter of a

few hours in the case of storage rots and some bacterial diseases, or very long, as in the case of wheat bunt, extending from harvest time, when the spores come to rest in the brush or crease of a healthy seed, through the dormant period of storage during the summer or winter on through the period of infection of the seedling and the development of the wheat plant, until the first symptoms of the bunt disease appear, nearly a year after inoculation took place.

But although symptoms have not yet appeared, the pathogen is passing through stages of activity during the incubation period.

If the inoculum is a spore, germination must occur, and the germ tube or infection hypha must penetrate to the interior of the host plant, and establish feeding mycelium. These processes vary greatly with different pathogens and hosts. Host plants are protected by a variety of chemical and mechanical barriers (cuticle, cork, etc.) through which the infection thread must pass. Some pathogens make use of natural openings in penetrating the host, stomata, lenticels, water pores, and glands, others, such as the soft rot bacteria, depend on scratches, insect stings, and other wounds, for entering the host tissues, while the most aggressive parasites force their way in through the uninjured cuticle by mechanical pressure or by enzymes that dissolve the protective layers of the host. Fig. 4 illustrates this method of forceful penetration, the infection thread expanding to form a disklike sucker or appressorium that attaches the hypha to the host, and through which passes a needlelike peg that forces its way down through the cuticle, and then broadens out as a feeding hypha.

The processes of germination and penetration are highly dependent upon external factors, especially moisture and temperature, and in some cases the host itself secretes attractive or stimulatory chemicals without which the pathogen would be unable to infect. Some fungus spores will not germinate except in the presence of an extract of host tissues.

THE INFECTION STAGE. The period during which the host responds, symptoms appear, and the disease develops is called the infection stage. It extends from the first response of the host until its final reaction to the pathogen. It may be very short, as in brown rot of peaches, potato late blight, or soft rot of vegetables, in which cases destruction can be complete within a few days, or it may extend for periods up to several years as in the wood diseases of trees. During the infection stage, lesions of disease appear and enlarge, secondary symptoms follow, the pathogen multiplies and begins asexual reproduction. This is the active period of infection and if death does not occur it is usually followed by a decline in activity; the lesions cease to enlarge and the host either recovers or becomes invaded by secondary organisms that further its destruction. The

decline in activity may be due to healing processes in the host (as walling off the lesions by cork or periderm), to a change in the environment so that the disease is no longer favored, or to the accumulation of waste by-products of the parasite that restrict its further development. Under the influence of alternating temperatures or light intensities, periods of high activity may occur intermittently, producing zonate, target-board, or "frog-eye" types of lesions.

In the invasion of new tissues the pathogen may be intercellular, feeding upon materials that are present or released between the cells or, in the case of some fungi, such as rusts and powdery mildews, the intercellular mycelium may send absorbing organisms (haustoria) within the living cells as shown in Fig. 4. Some pathogens, such as the clubroot organism, live entirely within the host cells.

Pathogens often excrete powerful enzymes that break down the plant constituents and render them available for nutrition of the pathogen, cellulase which breaks down cellulose, lignase, pectinase, proteinase, and many others. Or potent poisons may diffuse outward from the pathogen, killing cells in advance of its progress, so that the pathogen follows as a scavenger, feeding on the dead and dying cells that have been destroyed by its toxins. This may lead to a response in the plant far in advance of the area occupied by the pathogen.

2. **SAPROGENESIS** is the period during which the pathogen is no longer associated with the living host. The pathogen may be actively feeding and developing, living a saprophytic existence, largely on the decomposing remains of the host, as in the case of apple scab and numerous other diseases in which the sexual development occurs on the debris of the crop, or it may be largely or entirely dormant in the period of saprogenesis. The pathogen often is dependent on the residue of its natural host for its saprophytic existence, which in part explains the value of rotation in controlling plant diseases. Pathogenesis may be a fixed and more or less necessary part of the life cycle (apple scab, cereal scab) or may be only rare and incidental to a saprophytic existence. Thus, *Rhizopus nigricans*, the black bread mold, ordinarily is a saprophyte but readily parasitizes sweet potatoes and strawberries in storage. Saprogenesis is lacking in nature in the case of obligate parasites such as rusts, powdery mildews, and viruses, while with other pathogens, such as the smuts, potato late blight fungus, and the bacteria of fire blight and crown gall, saprogenesis is wanting in nature but may be experimentally produced on culture media in the laboratory.

Epiphytology

Despite the presence of a pathogen, its efficient dissemination, and a susceptible host, plant diseases rarely "make the headlines" by becoming

so disastrous that there is enormous crop loss, suffering, or even famine. But occasionally this does occur and the disease is then said to be *epiphytotic* (*epi-phyton* = "among plants," corresponding to *epidemic* in human disease or *epizootic* in animal disease). In contrast, diseases such as the wilts of tomato, flax, and watermelon, which are always present in a locality in relatively uniform amount, are called *enphytotic* (cf. *endemic*, *enzoötic*). A disease is called *sporadic* if it occurs only occasionally and does not involve a large number of individuals, as in the case of root knot in the northern states, where it is relatively unimportant in most years and locations, but occasionally becomes locally prevalent.

Various combinations of circumstances may produce epiphytotics, as is shown in the following examples.

Epiphytotics Due Primarily to Weather Conditions: **STEM RUST OF WHEAT.** This is normally enphytotic, but occasionally it has broken out in epiphytotic proportions, the 1935 outbreak destroying one-fourth of the American crop. The factors that conspire to produce this destructiveness include the following:

- a. A mild winter that permits the rust fungus to overwinter abundantly in the growing wheat of Texas. The milder the winter, the farther north in Texas will this occur.
- b. Moderately cool temperatures during the growing season, especially during the period from emergence of the heads to full bloom.
- c. Persistently humid, dewy, misty, or foggy weather during this period.
- d. Extension of these temperature and moisture conditions over a wide wheat-growing area.
- e. A large acreage of susceptible wheat.
- f. A period of hot dry weather just before harvest. This aggravates the water-loss due to rust.

Epiphytotics Due Primarily to Unnatural Culture. Many diseases that are relatively unimportant in the mixed plant associations of nature, become serious when the host is grown in an unnatural manner by the planting of large areas with a single susceptible species of plant. An example is:

PHACIDIUM BLIGHT OF CONIFERS. This is a fungus disease of young trees that spreads directly from plant to plant under the snow. It is native in the maritime provinces of Canada where it has been unimportant since in a native forest young susceptible trees are widely scattered and do not afford extensive contact with one another. With the growing drain on northern forests for the paper industry, reforestation became necessary, and for this purpose, vast nurseries of coniferous seedlings were estab-

lished. Under these unnatural conditions, *Phacidium* blight ran rampant through the nurseries, destroying great quantities of the young trees. Ultimately the disease has become controlled by an application of lime-sulfur spray before snowfall.

Other diseases which were unimportant until the host was cultivated in huge acreages are the downy mildew or "blue mold" and the white rust of spinach in Texas.

Epiphytotics Due Primarily to the Introduction of Disease in Some Part of the World Where It Has Not Previously Existed:

LATE BLIGHT OF IRISH POTATOES. About 250 years after the potato had been introduced into Europe, the blight fungus became destructive in European potato fields. The native home of the host appears to be the Andes Mountains of South America, but there the native potatoes show no material tolerance for the fungus and it is believed that the disease is native to Mexico where most of the wild potato species are resistant to or immune from blight. During the centuries of domestication and breeding of the potato in Europe, whatever tolerance of the fungus it may have had disappeared, and when the fungus was introduced, presumably on some solanaceous plant from Mexico, it came into contact with extensive plantings of a highly susceptible host. The inevitable result was the great epiphytotic of 1843-1845 with famine and migration following in its train. (See p. 3.)

CHESTNUT BLIGHT. The American chestnut had never been exposed to its blight pathogen when the fungus was brought from the Orient on infected nursery stock. In China, its native home, the chestnut is relatively resistant to the blight fungus, which there causes an inconsequential disease of the chestnut. The American chestnut proved highly susceptible and the epiphytotic beginning in 1904 advanced without hindrance until it has practically exterminated the chestnut from its extensive North American range.

DUTCH ELM DISEASE. This also appears to be native in Asia, if one may judge by the resistance of the elms in that part of the world. It appeared in Europe about 1922 when it soon produced such a destructive epiphytotic on the highly susceptible European elm that great areas were depleted of this species. About 1930 or earlier it was brought to America in shipments of elm burls (galls of beautiful grain, used for cabinet making). From New York it spread out radially into Connecticut, Pennsylvania, and New Jersey, destroying many thousands of trees. The American elm epiphytotic differs from that of the chestnut blight in that the well-established Bureau of Plant Quarantine was able to initiate an extensive inspection and eradication program at the outset, and this epiphytotic has not swept with unimpeded force through the range of the American elm.



Time in years. The height of the curve indicates destructiveness.

FIG. 207. The grand cycle of disease. Explanation in the text.

Epiphytotics Resulting from Plant Breeding. Breeding to improve crops is not without its dangers, although these are greatly outweighed by its advantages. As new combinations of hereditary factors are produced by the breeder, some of these may prove susceptible to previously unimportant plant pests, with disastrous results. This was the case of the Victoria-type oats, developed for resistance to rusts and smuts, which fell victim to a fungus that formerly had been a minor pest of grasses, as described on p. 180.

The Grand Cycle of Disease. When a disease is first introduced into a new area it may rapidly increase to epiphytotic proportions. Unless the host species is completely wiped out, in time the crest of the epiphytotic is passed and the disease subsides to a moderately low level and continues to be enphytotic except as this is interrupted now and then by conditions that favor a transient epiphytotic. Among such favoring conditions are weather, the introduction of a new host, the development of a particularly aggressive strain of the pathogen, or abnormal methods of culture. This sequence of events, diagrammed in Fig. 207, constitutes the grand cycle of disease.

After the height of the epiphytotic is passed the recession of the disease is due to the following four factors:

REDUCTION IN THE POPULATION OF AVAILABLE HOST PLANTS. This may be due partly to destruction of hosts by the disease, partly to changes in cropping practices with shifts to other types of crops.

THE DEVELOPMENT OF RESISTANT POPULATIONS. During the epiphytotic, thousands or millions of the most susceptible individuals succumb. A few of those having some natural resistance may survive and propagate their kind, the more resistant individuals surviving each generation until the resistance has become ingrained in the species.

Man can speed up this developing resistance in the population: (1) by painstaking search for resistant survivors, protecting them from loss due to other natural hazards, and aiding in their propagation under conditions that subject them to repeated exposure to the pathogen and thus foster natural selection of the more resistant individuals of each generation; (2)

by introducing into the epiphytotic new genetic types secured through hybridization of resistant species with commercial or native varieties, thus increasing the numbers and types of survivors in the epiphytotic; (3) by a combination of these two practices, and this is the background of man's greatest successes in combating epiphytotics.

ARTIFICIAL CONTROL. Man sometimes has been able to check epiphytotics by wide-scale use of cultural and regulatory control measures. Examples are the checking of late blight by Bordeaux spraying in the last century, reduction in attack of certain peach viruses by an eradication and quarantine program, abatement of violet root rot of alfalfa by a shift to a shorter rotation cycle with this crop, and reduction of damage from squash foot rot by general use of old seed, from which the causal organism has died out.

NATURAL CONTROL. The recession of an epiphytotic may be due in some measure to increasing difficulty on the part of the pathogen to maintain itself in the presence of antagonistic microorganisms.

HYPERPARASITISM. Some plant pathogens are in turn parasitized by other organisms (*hyperparasitism*). For example, the blister rust fungus is less destructive in Europe than America, because in Europe there is a hyperparasitic imperfect fungus, *Tuberculina maxima*, which feeds on the blister rust spores and destroys them. Similarly, the cereal rust fungi are hyperparasitized by the imperfect fungus *Darluca filum*, a parasite on the pustules. Plant parasitic nematodes frequently are attacked by other predatory nematodes or by nematode-trapping fungi (see Fig. 181). Even pathogenic bacteria are subject to parasitism by bacteriophage which has been shown to be active in destroying colonies of the bacterial pathogens in crown gall and wilt of corn.

ANTIBIOSIS (COMPETITION). In other cases a pathogen is held in check by its struggle to secure food, grow, and reproduce in the presence of other organisms competing for the same food or space. This is true particularly when a pathogen enters saprogenesis and finds itself in competition with true saprophytes that may be better adapted to saprophytic life than the pathogen. The abnormal abundance of a pathogen during an epiphytotic cannot long be maintained in the face of natural competition with other organisms, and ultimately these will bring about a reduction in the pathogenic population and thus contribute to the recession of the epiphytotic. In a very limited way, man sometimes can put antibiosis to good use as in the control of Texas root rot, root knot, and wheat foot rot, by soil amendments of organic materials that encourage the growth of soil saprophytes and hyperparasites at the expense of the pathogens (see Figs. 80 and 81).

Origin of Plant Diseases. The questions are often asked: Why are new plant diseases continually appearing; where are they coming from; why are there so many more diseases now than years ago? The principal reasons are five, as follows:

WIDER DISTRIBUTION OF OLD DISEASES. The increased introduction of diseases into new localities, where they are viewed as "new diseases," is due largely to the progressive broadening of plant commerce. More facile and varied means of transportation, the use of refrigeration, the demands for exotic and unseasonable fruits, vegetables, and ornamentals consequent on improved standards of living, and the tendency toward larger and larger units of agricultural production and marketing, have all conspired to lift the production of nursery stock, plant foodstuffs, flowers, and seed out of local ventures to satisfy local demands into undertakings planned to meet national or even international demands. As the products have entered new areas their diseases have accompanied them.

INCREASING PROPORTION OF SUSCEPTIBLE HOSTS IN THE POPULATION. Horticulturists, agronomists, and plant breeders are striving constantly to develop new selections or hybrids with more desirable esthetic or utilitarian qualities. In selection or breeding for these qualities it is easy at the same time to lose other qualities, among them the resistance to disease characteristic of the ancestral species. This loss of resistance with improvement along other lines is characteristic especially of vegetatively propagated crops that have been in domestication for many centuries, such as the potato, and crops like wheat, oats, and sorghum in which the better varieties have resulted from extensive synthetic breeding. With the loss in resistance from this cause, there follow the ravages of diseases to which the ancestral species were tolerant or resistant.

The extensive use of hybrid corn illustrates another danger. The uniformity of hybrid corn, while an advantage agronomically, is a potential hazard pathologically. Natural populations of hosts usually contain individuals with varying degrees of resistance to a given pathogen. The agronomic variation seen in individuals in a field of open-pollinated corn is accompanied by variation in disease susceptibility. Disease cannot sweep through heterogeneous populations with the same freedom as in fields in which every individual is almost exactly as susceptible as every other one. When, as in the hybrid corn program, we plant vast populations of very similar plants, we are creating the possibility for totally effective epiphytotics should there appear virulent pathogens to which the hybrids are not resistant.

INTENSIFICATION OF AGRICULTURE. As civilization progresses and populations increase, demands for food are greater and it becomes neces-

sary to produce more on less land. This leads to intensified agriculture, such as characterizes the market garden areas around cities or the agriculture of European countries. A disease causing a 5 per cent loss in a field crop with a value of \$50.00 per acre (\$2.50 loss) might be considered negligible or too small to warrant the expense of control measures. It might even be cheaper to offset this loss by increasing the acreage by 5 per cent than to go to the expense of controlling it. With intensified agriculture the value of the crop might be \$500.00 and of the loss \$25.00 or even much more, since the plants are grown closer together, and well watered, which could materially increase the percentage of loss. Under these conditions the disease assumes importance which it did not have previously; it might even be regarded as a "new disease."

IMPROVED RECOGNITION OF DISEASES. Plants in a field become yellow or wilt and die. Forty years ago this would probably have been attributed to drought, poor soil, alkali spots, or unfavorable weather. At that time there were few trained men who could identify the cause of the trouble; plant pathology was just coming into being. Today the yellowed plants are examined and the trouble is demonstrated to be due to a virus disease, root decay, or a vascular infection. We may call it a "new disease," but it is new only to science; it may have been present for hundreds of years but never recognized as a contagious disease.

AUTHENTIC NEW DISEASES. Nearly all so-called "new diseases" are of the preceding types. Rarely a disease may not only be new to science but actually new to nature. This may be due to the production, by hybridization or mutation, of new physiologic races of pathogens or strains with exceptionally high virulence, transforming a benign and relatively harmless disease into a dangerous one. Or a pathogen on a wild host may produce a strain with a wider host range capable of passing to a cultivated crop and producing serious disease. This would be aided by man's introduction of new crops in areas where they had not been grown previously.

Prediction of Epiphytotics. If it were possible to predict epiphytotic outbreaks of plant disease in sufficient time to allow intervention of precautionary measures, enormous losses might be averted. Because of the many requirements which must be met before a disease can become epiphytotic, in particular the unpredictable weather, there are few diseases with which such predictions are possible. There are notable exceptions, however, particularly in cases in which the majority of antecedent requirements are met regularly and where one or two observable antecedent circumstances will turn the balance in favor of epiphytotic outbreaks. As examples we have the cases of:

BACTERIAL WILT (STEWART'S DISEASE) OF SWEET CORN. The corn wilt bacteria overwinter chiefly in hibernating flea beetles, the survival of which depends on mild winter temperatures. For several years accurate forecasts of wilt severity based on December–February temperatures have been issued before corn planting time, permitting evasion of the disease by a change in planting plans.

KEEPING QUALITY OF CRANBERRIES. The cranberry crop, harvested in the fall, must be stored until the principal times of marketing—Thanksgiving and Christmas. The storage requirements differ with the keeping quality of the berries. Forecasts of keeping quality based on weather during the growing season have been quite regularly accurate and have made it possible to store the crop most safely and economically.

SMUTS OF THE BLOSSOM-INFECTION TYPE. In these diseases, such as loose smut of wheat, infection occurs a year before symptoms appear and several months before planting time. The amount of infection depends largely on the amount of inoculum in the field and moderate moisture at blossom time. A study of the field conditions at that time gives a sound basis for predicting loose smut incidence a year in advance, in ample time for the use of the preventive hot water seed treatment.

APPLE SCAB. Here the primary inoculum consists of ascospores discharged from overwintered leaves. Examination of these leaves in the spring to determine the moment of ascospore discharge coupled with an analysis of existing temperature and moisture conditions, serves as the basis for radio spray warnings in certain apple-growing areas. These warnings result in the most effective spray programs.

POTATO LATE BLIGHT. Forecasts of late blight epiphytotics in time to permit growers to protect the crop with sprays, based on a study of weather and blight development in scattered observation plots, are discussed on p. 217. Downy mildews of cucurbits and tobacco are also being forecast.

WHEAT LEAF RUST. An accurate forecast of rust is useful to the grower who has only a fair stand of winter wheat, which would better be replaced by a summer crop if a rust epiphytotic is threatening, but which might yield a profitable harvest in case of freedom from rust damage. In the Southwest, leaf rust damage in April–June is correlated with weather and rust development in February and March, and for several years accurate rust predictions have been made on April 1, in time for planting corn, sorghum, cotton, or summer legumes when a rust epiphytotic is predicted.

CURLY TOP OF SUGAR BEET. The earlier and larger the migration of beet leaf hoppers from winter host plants into beet fields, the greater will be the damage from curly top. In the southern sugar beet areas a single

survey of host conditions and leaf hopper numbers in January indicates the probable severity of damage to the beets in May or June.

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Chapter 18

Principles and Procedures in the Control of Plant Diseases; Control of Plant Diseases by Regulation

The object of plant disease control is to prevent economic loss and increase the value of the crop, to combat pathogens only insofar as they are producers of crop loss. Control practices are desirable only when the cost, in money and effort, is materially less than the loss from disease. Sometimes it is neither desirable nor possible to exterminate the pathogen, the function of control being to reduce the losses to a low level at the least expense.

Disease control is but one of many considerations in growing crops. Control measures must be planned to fit into the program of crop production so that the program as a whole will give the greatest returns. Whenever possible this program should include control of several diseases or diseases and insects in one operation. If control measures are opposed to other desirable practices, a compromise should be worked out to give the greatest return, all things considered. For example, destruction of infested crop debris often is helpful in disease control, but if this robs the soil of needed organic matter the benefit from burning the refuse may be less than the benefit from plowing it under. Early plowing after harvest often reduces disease in the next crop, but if this exposes the soil to severe erosion, the loss may be greater than that caused by the disease.

Disease control practices must be worked out as individual problems in each location. The best control methods will differ from one farm or area to another, according to weather, site, soil, type and condition of the crop, methods of culture, and value of the crop. In all cases control should have clearcut objectives. Spraying, dusting, or any other control practice without a concrete purpose is wasteful and may even be harmful to the crop.

Control Procedures. When a plant disease threatens a crop, the four following procedures may be followed:

EXCLUDING THE DISEASE BY REGULATION through the use of embargoes or quarantines enforced by an inspection service empowered to disinfect,

eradicate, or condemn diseased plants or plant materials to prevent them from becoming sources of infection in disease-free areas.

THE DEVELOPMENT AND USE OF DISEASE-RESISTANT CROP VARIETIES. (See Chapter 19.)

CULTURAL PRACTICES THAT ENABLE PLANTS TO ESCAPE OR RESIST DISEASE, the term being used in its broadest sense to include the use of noninfested or disinfested seed and soil, desirable methods of caring for the crop, the removal of undesirable plants, and the protection of the crop with sprays and dusts. (See Chapter 20.)

ABANDONMENT OF THE CROP may be the only recourse if the disease is serious and all other methods fail. This has been the case in the past with the *Fusarium* wilts of banana, flax, and watermelon, and even today there are some growers of watermelons who abandon infested land for this crop in preference to planting wilt-resistant varieties. Extensive acreages of sugar beets and sugar cane were abandoned before the development of virus disease-resistant varieties of these crops.

Control of Plant Diseases by Regulation

In nature, plants are in equilibrium with pathogens and insects; through centuries of exposure the more susceptible individuals have succumbed, and the surviving species are usually highly tolerant of their pests. Man has upset this equilibrium by introducing diseases to new localities and producing new, but unfortunately more susceptible types of crops. The consequence has been disastrous plagues of disease and insects. Although the damage had been done in the case of the chestnut blight, the irreparable loss from that introduced disease was a strong element leading to the passing in 1912 of the National Plant Quarantine Act, at that time directed against the dangers of pine blister rust, potato wart, and the Mediterranean fruit fly. Quarantines for protection against other diseases and insects soon followed, until today there are 22 foreign and 17 domestic federal plant quarantines or restrictive orders in force. Meanwhile, the individual states were becoming more active in promulgating quarantines restricting the movement of diseased or infested plant materials from one state to another so that by 1944 there were about 247 state quarantines directed at 42 insect pests and 28 plant diseases.

Justification for attempts at protecting agriculture from introduction of foreign diseases is seen in the long list of important diseases known to have entered America from abroad, including flax rust, apple scab, potato blackleg, tomato leaf spot, pine blister rust, melon mildew, cherry leaf spot, peach leaf curl, the Dutch elm disease, and wheat stripe rust. Many other destructive plant disease organisms exist abroad, awaiting introduction to America.

BIOLOGICAL ASPECTS OF QUARANTINES

For a quarantine to be effective it must be intelligently devised and adequately enforced. Intelligent legal control requires knowledge of:

1. The nature of the pest or disease, its life history, and particularly its means of dispersal. Is it spore-borne? How resistant are the spores to cold, heat, drying, and aging? Is it wind- or insect-borne or transmitted mainly by man? Is it spread with seed or through vegetative reproduction, or in agricultural products, or only in living plants? Is it restricted to one or few cultivated hosts or is it widespread on native plants?

2. The present distribution of the pest or disease. Is it already established in the area to be protected or in an adjacent area with no natural barrier between? Here we must give attention to the distribution of physiologic races of plant pathogens. A disease may be established in the country but the results of introduction of a new, foreign race of the pathogen may lead to the failure of formerly disease-resistant varieties acting in the same fashion as the introduction of an entirely new disease. It is known, for example, that in Peru there is a particularly virulent race of stem rust which could cause havoc if introduced into North America.

3. The probability that the pest or disease will be introduced in the absence of regulation. Only where a pest or disease is carried by man in plant materials is there any ground for attempted control by regulation. For example, no useful end would be served by restricting the movement of seed from areas of Texas root rot infestation, because the disease is not carried by this means.

4. The probability that the pest or disease will become established and important if introduced. Many cases might be cited of diseases which remain within a limited area despite countless chance introductions into new areas. The distribution of Texas root rot, for example, has undergone no essential change for 50 years or more, yet infested plants and soil have doubtless been transported to new areas many times. Even when root rot-infested soil is deliberately transplanted for experimental purposes under the most favorable circumstances, it is difficult or impossible to establish the disease in the new location. If there exists satisfactory evidence that the disease could not establish itself in a new area, because of unsuitable environmental or other causes, a quarantine against the disease imposes needless barriers to trade, is wasteful in its enforcement, and accomplishes no useful end.

5. The probability that the quarantine will effectively prevent or delay the introduction of the pest or disease into protected areas. Where man is the principal agent of dispersal and the activities of man can be regulated, a quarantine has promise of proving

effective. But many pests and diseases are spread by other agencies, particularly wind. In such cases, only if the infested and protected areas happen to be separated by natural, geographical barriers such as mountains, oceans, and deserts is a quarantine likely to succeed.

While the need for a complete knowledge of the biological basis for a quarantine is obvious, there are cases in which it would be disastrous to wait until this knowledge is available before undertaking the quarantine. In such cases a temporary quarantine may be justified as an emergency measure. Any quarantine, and above all an emergency quarantine of this sort should be reconsidered periodically and modified as the pathologic or economic picture changes, or it should be rescinded entirely once the danger or the cause for the quarantine no longer exists.

ECONOMIC ASPECTS OF QUARANTINES

Any quarantine is a restriction of trade. Even if it is intelligently conceived and effectively enforced, so that needed pest control is accomplished, there are bound to be individual growers and buyers of produce who will suffer economic loss from such a quarantine. If, as is sometimes the case, a quarantine is enacted primarily as a trade barrier for the purpose of protecting a local industry, or as a retaliatory act of one state or country against another, disguised as a pest control measure, except for the protected minority, the gross economic loss may be even greater, besides arousing prejudice against legitimate disease regulation.

In any legitimate quarantine, part of the gain resulting from pest control is offset by the lost markets of the growers within the quarantined area and the expense of enforcing the quarantine. For the net result to be beneficial to the people as a whole, the gain from such means of pest control must be an important one, i.e., the pest must be a serious menace to crops, and the loss in markets and cost of enforcement must not be so great as to overbalance the gains. This clearly means that quarantines frequently must represent compromises with some sacrifice of the efficiency of the quarantine to avoid undue hardship on the grower and the consumer.

The more important items on the balance sheet of a quarantine measure are given in Table 7.

Each of these items is important in itself and may have far-reaching secondary effects. It goes without saying that the economic repercussions of a quarantine are so forceful that no permanent quarantine should be enacted until a thorough analysis, both biological and economic, by competent and unprejudiced authorities, establishes the basis for quarantine laws that will accomplish the desired purpose with the least possible hardship to growers and consumers.

Table 7
BALANCE SHEET OF QUARANTINES

<i>Debit</i>	<i>Credit</i>
Cost of enforcement.	Freedom from losses due to the depredations of the pest (which may be partly offset by higher prices for the reduced crop if no quarantine is employed).
Loss of markets.	Freedom from the cost of direct control measures against the pest.
Condemnation and abandonment of crops (which may be partly offset by government reimbursement in some cases).	Trade advantage of growers outside the quarantined area.
Waste in adjustment to new types of agriculture.	
Losses from retaliatory measures.	

Regulation of foreign plant diseases often has important international aspects. International coöperation may be necessary when one considers, for example, that introduction of a destructive disease in Canada may be as dangerous to the United States as if the disease were brought directly to this country.

MECHANISM OF PLANT QUARANTINES

If a quarantine law is worth having, it is worth enforcing, and quarantines which cannot be adequately and effectively enforced should never be enacted. The method of enforcement varies with the disease problem and the methods of trade.

1. Embargoes. An embargo prohibits any movement of susceptible or affected plant materials from a quarantined area into protected areas. An example is the present United States embargo against Australian wheat on account of the danger of introducing flag smut.

2. Inspection at the Point of Destination. Many plant propagating materials entering the United States are inspected regularly at the customs ports and allowed entry only after having been declared free of injurious insects and diseases.

3. Inspection and Certification at the Point of Origin. Certain plant propagation materials from abroad, such as bulbs, may enter the United States if a permit is secured certifying that the materials were inspected in the shipping country and found free of injurious insects or diseases. Inspection and certification at the point of origin is the basis of interstate shipments of certified seed and nursery stock and to be fully effective should include inspection of the seed crop or stock while growing (Fig. 208).

4. Disinfestation of entering plant materials may be required either at the point of origin or at the port of entry. This applies, for example, to citrus seeds and some bulbs from abroad.



FIG. 208. The function of nursery inspection is providing growers with healthy nursery stock. (*Left*) A state inspector examines a planting with the owner, pointing out the stock that is sound and suitable for sale, and that which should be culled. (*Right*) A pile of cull apple trees. Conscientious and careful culling, aided by state inspection, is a regular part of good nursery practice. (Courtesy, Okla. State Board of Agriculture.)

5. Special Permits. Plants and plant products for scientific and breeding work, botanical specimens, and exhibit plant material may be brought into the United States under special permit, even though a quarantine prevents commercial shipments of the product.

6. Unrestricted Shipment. Where no important pest is involved, plant materials of importance in world trade may be shipped from one country to another without disinfestation, or other restrictions, although they are subject to occasional inspection.

These regulations apply to international shipments, and many similar regulations govern the shipment of plant products from one state to another, under either state or federal law.

PRESENT-DAY QUARANTINES

The following are among the more important quarantines at present in effect:

1. Federal Quarantines Affecting International Trade:

EMBARGOES OR SPECIAL QUARANTINES GOVERN THE IMPORTATION OF: pines and gooseberries from all countries (blister rust), Irish potatoes from all countries but Canada (potato wart), cottonseed and certain cotton products from all countries, certain packing and wrapping materials from all countries, sugar cane, alligator pears, citrus nursery stock, raw corn and related grains, sweet potatoes and yams, banana plants, bamboo propagation materials, seed rice, wheat from many countries, and raw sorghums.

ENTRY UNDER PERMIT WITH INSPECTION, and, as provided, disinfestation, governs the entry of flower bulbs, propagating materials of fruit and nut crops, tree seeds, rose propagating materials, and seeds of woody and perennial ornamentals.

2. Federal quarantines affecting interstate trade have to do with diseases or insects that are established in some parts of the United



FIG. 209. Application of common salt in eradicating barberry, alternate host of the stem rust. Ten pounds is sufficient to kill a bush of average size with a diameter of about 12 in. at the base. (Courtesy, Col. Agr. Exp. Sta.)

States but as yet have not spread to other parts. The principal insects concerned are the *gypsy and brown tail moths* in New England, the *Japanese beetle* in the eastern states, the *cotton bollworm and Thurberia weevil* in the South and the *Mediterranean fruit fly* in Florida. The diseases involved are *white pine blister rust* (no interstate movement of five-needle pines or currants and gooseberries), *stem rust* (no movement of susceptible barberry bushes into the protected northern middlewest section), and the *Woodgate rust* (no movement of susceptible pines from New York State). The quarantines are augmented by extensive eradication programs in the cases of stem rust, and pine blister rust (Fig. 209).

3. State-imposed Quarantines Affecting Interstate Trade.

Quarantines of the state departments of agriculture are aimed at excluding from a state those insects and diseases that occur in other states and might be seriously damaging if introduced. The state quarantine laws vary so widely from one state to another that a serious problem faces the interstate shipper. Large seed concerns and nurserymen sometimes must maintain a special organization to handle the tagging and permits required for meeting all of the various state quarantine regulations. The problem of state quarantines is further complicated by the common practice of

retaliatory quarantines and fees on the principle that "if you tax us, we'll tax you, and if you quarantine our crops, we'll quarantine yours." Attempts are being made to encourage the states to adopt more uniform quarantine laws based on sound biological and economic principles.

While it is beyond the scope of this book to discuss in detail the various state quarantines, these may be summarized as follows:

The number of state quarantines varies greatly. The states deriving some protection from the Rocky Mountains have the greatest number: Oregon, 13; California, 21; Washington, 15; Idaho, 19; and Arizona, 16. This may be justified on the grounds that these states are free from a number of diseases and insects occurring east of the mountains, while mountains afford a natural barrier to aid in disease control by regulation. Other states have 11 quarantines or less, with the fewest in the North Atlantic states and the northern Great Plains and Corn Belt states.

The following are the leading plant diseases subject to state quarantines: pine blister rust, 17 quarantines; phony peach, 11; peach mosaic, 9; cereal stem rust, 8; virus diseases of cane fruits, 7; chestnut blight, 5; citrus diseases, 5; and peach yellows, rosette, and little peach, 5. Other plant diseases which are subjects of plant quarantines in four or fewer states are crown gall, brown rot of stone fruits, Pierce's disease of grapes, X-disease of peach, strawberry red stele, cranberry false blossom, bulb nematodes, lily diseases, azalea flower spot, Dutch elm disease, filbert blight, cedar-apple rust, Texas root rot, hop mildew, potato nematode and wart, and root knot of tomato.

STATE CERTIFIED SEED. Many of the states maintain Crop Improvement Associations, or other similar agencies delegated to oversee the production of disease-free certified seed of many crops. While this may not be directly governed by state law and may have no disciplinary power beyond allowing or disallowing certification, its effect is similar to that of other regulatory measures in aiding to keep disease-infested seed out of commerce. Other states recognize this, and certified seed is exempt from some of the restrictions imposed by law on ordinary seed.

EFFICACY OF QUARANTINES

Authorities disagree as to the value of quarantines. On the one hand there are those who have watched the ceaseless spread of chestnut blight, the gypsy moth, boll weevil, Japanese beetle, and stem rust, despite costly quarantines enacted to check their spread.

There is reason to feel disappointed in the results of quarantines in some cases. On the other hand, however, there are well-authenticated instances in which significant and important pest control has resulted from quarantines. The notorious wart disease of potatoes was stamped out

of Sweden and Canada and kept under control in Pennsylvania by means of vigorous and prompt quarantines. Corn smut has been eliminated from Australia. With the Dutch elm disease and peach mosaic, combined quarantines and eradication have been followed by the finding of smaller and smaller numbers of diseased trees each year after the program got under way. Citrus canker, a bacterial disease that was discovered in 1915, seriously endangered Florida citrus growing. Combined federal, state, and private efforts in a \$2,500,000 eradication campaign were successful in stamping it out, as has also been done in South Africa and northern Australia. Other quarantines have not prevented the spread of plant pests, but have slowed it down and this has been a benefit in giving growers in the path of the spread time to readjust their systems of agriculture without serious losses.

Usually, regulatory measures must be compromises, consideration being given to the economic and political results of trade limitation by law. As such they cannot be expected to be completely effective in preventing spread of plant diseases, and if they protect plants even partially and temporarily they may be of distinct benefit.

The truth probably is intermediate between the more extreme views of the attackers and the defenders of quarantines. Some quarantines have been costly and useless, others have been abused and used as purely economic measures, but in numerous cases where regulatory laws have been skillfully and intelligently devised and adequately enforced, the protection against plant pests has well repaid the cost and effort expended.

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- , Notice of Quarantine 37, Reprinted 1936, Nursery stock, plant and seed quarantine.
- You should also examine copies of the quarantine and certification regulations of your own and neighboring states, usually available from the State Board of Agriculture, Agricultural College, or State Agricultural Experiment Station.

Chapter 19

Control of Plant Disease by Inducing Resistance

Economic Significance of the Development of Disease-resistant Crop Varieties

The pioneer in breeding plants for disease resistance, W. A. Orton, has pointed out that, "Nature has been breeding disease-resistant plants since the world began." When a population of plants is exposed to the ravages of a killing pathogen, natural selection for disease resistance begins to operate. The more susceptible genotypes are destroyed. Only those individuals that possess some degree of resistance survive and reproduce, transmitting their semi-resistance to their progeny. These in turn are again exposed to the disease which again culls out the most susceptible types, and thus in each succeeding generation the level of resistance is raised until relatively nondestructive equilibrium between host and pathogen is reached.

In nature this is a slow and gradual process. Plant breeders and pathologists can greatly accelerate the process by selecting, caring for, and propagating the more resistant individuals of a large population, by hybridizing plants and producing a greater variety of genotypes on which natural or human selection can operate, and by exposing the plant populations to such extreme disease attack that the great bulk of nonresistant individuals are soon destroyed.

The deliberate development of disease resistance in plants extends back only to about 1890, but in these few decades great advances have been made. Coons' estimates of 1937 show that disease-resistant varieties of 17 farm crops have added between 60 and 70 million dollars a year to the American farm income, that with a number of crops the resistant varieties yield a 25 or 50 per cent benefit to the grower, that more than half the acreage of a number of crops is planted with resistant varieties, and that in certain crops, notably flax, sugar cane, and asparagus, practically the entire national acreage is planted with disease-resistant varieties. Some crops such as watermelons, cantaloupes, sugar beet, and flax could not be successfully grown today were it not for varieties resistant against

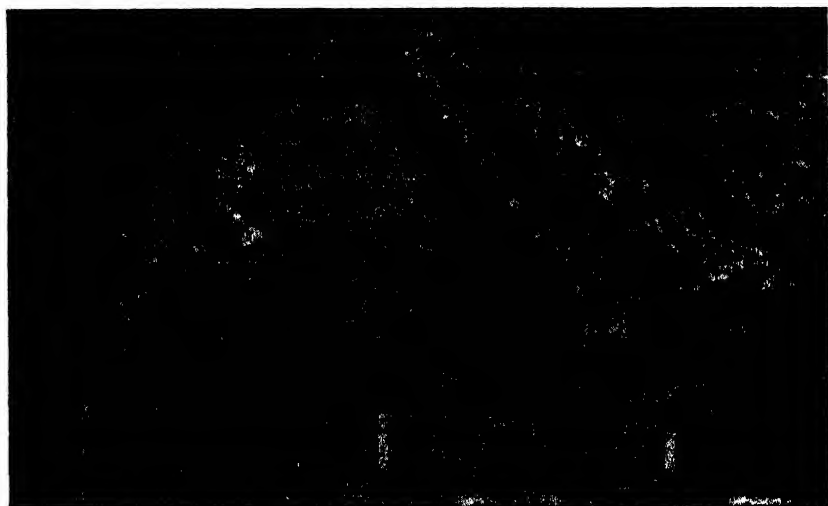


FIG. 210. The breeding of cabbage for resistance to *Fusarium* wilt ("yellows") is a notable example of achievement. In this experimental planting the center row is of a yellows-susceptible variety. The rows to left and right of it, and several other rows at the right are of resistant varieties. (Courtesy, O. A. Reinking, N. Y. Agr. Exp. Sta.)

their most destructive diseases. Few undertakings in science or industry have paid such huge dividends in comparison to the original cost.

The only cost of disease control through varietal resistance is the original cost of developing the varieties, and even though resistant varieties must be replaced by new ones from time to time, this is a decided advantage over the more costly practices of disease control by quarantines or by physical or chemical treatments. The economy, the labor saving, the ease of widespread adoption, the minimum of risk through errors, are the reasons why resistant varieties represent the best of all forms of disease control.

Some Basic Concepts of Plant Immunology

If we compare the attacks of a given pathogen on many varieties of a crop, we often find a graded series of reactions ranging from highly susceptible through intergrades to highly resistant. Resistance and its reverse, susceptibility, are relative, not absolute, terms. When we speak of some crop varieties as resistant and others as susceptible, we mean that the first have more resistance than the second. Immunity in a variety, that is, total freedom from a disease that attacks other varieties of the same crop, is rare.

Resistance may take several forms. A plant may be resistant to infection by a pathogen or the plant may permit infection but so limit the activities

of the pathogen that it cannot multiply freely or cannot cause lesions of sufficient extent to result in appreciable crop damage. If multiplication of the pathogen is prevented, as in resistant rust reactions, the pathogen cannot become widely disseminated, and the crop as a whole is relatively disease-free. If the lesions are small or if the plant tolerates the pathogen without marked interference with its vital activities, the quality and quantity of yield are not likely to be seriously reduced. We are interested in a practical degree of plant disease control, not in exterminating parasites.

The different degrees of susceptibility displayed by plants must not be regarded as fixed and absolute. They are profoundly modified by the environment. In preceding chapters many examples of plant diseases which are limited by temperature, light, moisture, host vigor, or other factors have been discussed. Often these environmental influences operate by affecting the susceptibility of the host plant, sometimes transforming a practically susceptible plant into a practically resistant one. The reactions of wheat and corn to *Giberella* seedling blight as influenced by soil temperature (see p. 413), the masking of rusts and viruses by high temperatures, and the relation of host vigor to wheat rust and foot rot, are examples.

Pathogens or their strains show different degrees of aggressiveness or virulence, ranging in series from relatively harmless ones to others that rapidly destroy plants. The intensity of disease is the product of the degree of virulence of the pathogenic strain and the degree of susceptibility of the host plant. A moderately resistant host may show considerable susceptibility if attacked by a highly virulent pathogen, and be quite resistant to the attack of a pathogen of low virulence.

The observed degree of susceptibility in any case is a product of many interacting factors of which the inherent susceptibility of the plant is only one. The degree of virulence of the pathogen, the age and condition of the plant, and the environment with its many effects on both host and pathogen, all must be suitable before susceptibility can be expressed. Development of disease has been compared with the operation of a complicated lock: every tooth and tumbler of the key and lock must be in proper alignment before the lock will open, and as the failure of a single correspondence between lock and key will entirely prevent the act of unlocking, just so the failure of any of the many factors required for disease expression will entirely inhibit the infection.

Considering the thousands of species of plants, each subject to a distinct few of the thousands of species of pathogens, it follows that any plant is resistant to most pathogens, any pathogen is unable to attack most host species, and combination of a pathogen with its proper host is rare

among the many combinations that cannot result in disease. An apple tree is bombarded with bacteria and spores of countless species of fungi, spores of corn smut, alfalfa rust, potato late blight, wood decay fungi, molds, and mildews. Not one spore in a thousand may be of a species of pathogen to which the tree is susceptible, and even in the rare case in which the spore is constitutionally capable of attacking the tree, it must fall in a suitable infection court, at a suitable time of year, and under suitable environmental conditions. For every spore that accomplishes infection there are millions that fail to do so, either because the conditions for infection are not fulfilled or because every plant is immune from or resistant to the vast majority of the pathogens with which the air is laden. Resistance or immunity is the law of nature, susceptibility the rare exception. Why is it that the alfalfa plant is susceptible to only one species of rust fungus? What is there about the plant which prevents the attack of any other of a thousand species of rusts? In a word, what is the mechanism or basis of resistance and immunity in plants?

Nature of Disease Resistance and Immunity

The armament of a plant against its enemy pathogens can be compared with that of a modern army. It includes mechanical defenses, each plant being a fortress enclosed within teguments of tough and chemically resistant cutin, lignin, cork, and cellulose, often bristling with spines or hairs, or coated with an impervious and repellent bloom of wax. The armament of the plant also provides chemical weapons of defense and offense: its inner economy may not include foodstuffs that are needed by the pathogen, or may include acids, alkaloids, tannins, and other substances that are toxic to the pathogen. Again, as in modern warfare, the plant may elude the pathogen to which it is susceptible by developing or functioning in such a way that susceptible and vulnerable infection courts are not available at the time the pathogen is active.

Mechanical Defenses: 1. EPIDERMIS AND CUTICLE represent the first-line mechanical defenses of plants. These vary in structure, and in some cases serve as a mechanism of protection. Barberry species vary, for example, in their reaction to stem rust, some being susceptible, others resistant. The resistant varieties are found to have a thicker epidermis than the susceptible ones.

2. STOMATA differ in size, number, and form in different plants, and this influences infection by pathogens that enter through them.

3. HAIRINESS of leaves or fruits is held to have some protective value.

4. WAXY COATING of stems in certain varieties of raspberries is correlated with their resistance to stem canker.

Chemical Defenses. Little is known of the chemical defenses of plants, providing a rich and fascinating field of discovery. We can envision the complexity of the situation by considering the rust fungi. Their need of a delicately adjusted substrate for development is shown in the fact that they are obligate parasites and must have living cells from which to obtain their nourishment. Not one of the thousands of nonliving media that have been tried will support their growth, and not just any living cell will suffice. The rusts are distinguished by their narrow host ranges, many of them attacking only one or two species of all they encounter in nature, or even developing on only a few varieties of a single species or, paradoxically, developing only on two widely different species of plants, as cedar and apple trees, or the fir tree and a single species of ferns. What is there lacking in all other kinds of plants, that is present only in the cedar and apple trees, or what combination of defenses protects all species of plants from this rust save only the wholly unrelated cedar and apple?

The physiologic specialization of the rust fungi gives further testimony of the delicate chemical adjustment between host and pathogen. There are more than 180 races of the variety of stem rust that affects wheat. These races are indistinguishable in appearance but each has its own peculiar conditions for life that differ from those of the other races, since each race finds itself able to develop on a group of wheat varieties different from the wheats that will support any of the other races. What is there about Ceres wheat that defends it against most of the common races of stem rust but permits race 56 to invade it? It is not mechanical defense, for the infection thread of any rust race enters resistant and susceptible wheats alike, develops a small mycelium and begins to draw nourishment from the wheat cells. But at this point the resistance of the resistant wheat expresses itself; the mycelium develops no further, and the rust dies.

Whatever these factors of resistance may be, they obviously relate to a complex chemical relationship between host cells and those of the pathogen, a chemical relationship so specific that it can occur not oftener than once in a thousand combinations of susceptible species and rust species.

It is easy to see why the subject of chemical defense in plants is still in its infancy. Of the few cases in which the details of the chemistry of resistance are known, Walker's experiments with the onion smudge fungus stand out as noteworthy.

J. C. Walker of Wisconsin first observed that the smudge fungus, *Colletotrichum circinans*, normally attacks only white onions, not those with red or yellow papery scales. But if he removed the colored scales the colored onions became susceptible to smudge. Acting on the assumption that the red or yellow pigments of the scales were in some way associated

with the resistance of the colored onions, he extracted the pigments with water and found that the spores of the fungus would not germinate normally, and that the fungus would not grow in extracts of the pigments, although it developed normally in similar extracts of white-scaled onions. The extracts of red and yellow pigments were analyzed by Link and Angell and the toxic substances found to be protocatechuic acid and catechol. It was found that these substances also protect the colored onions against certain other diseases.

Having studied the chemistry of disease resistance Brown and his students, in England, concluded that in the case of potato tubers attacked by certain fungi, susceptibility depends largely on the chemical action of the tuber in permitting the fungus to produce a type of pectinase with specific ability to digest the potato cell walls.

There is some indication that in the rusts the obligate parasitism and high degree of host specialization are related to the presence in plants of specific proteins which differ from plant to plant, as can be shown by serologic tests.

In other cases resistance has been ascribed to unsuitable food supplies or to inhibiting acids, oils, esters, and tannins in the host tissues, but the evidence in these cases is very limited.

Functional Defenses. Plants frequently avoid disease not by virtue of any defensive structure or product, but because they grow in such a way as to escape attack, even though quite susceptible. Early-maturing varieties commonly are used to avoid late-season diseases, such as early wheats which are not as severely attacked by rust as midseason and late varieties, or early cowpeas which avoid wilt and root knot. This is not true resistance, and if such early varieties are caused to mature later by late planting, they may be strongly attacked. Other forms of functional defenses include the habit of some plants of opening their stomata for only short periods of the day, or the development of an upright, well-ventilated plant body that avoids pockets of moist air in which pathogens might rapidly develop (correlated with gray mold escape in lettuce).

Another use of the principle of escape is seen in the practice of planting at excessive rates, on the assumption that even with a high mortality in the crop enough plants will survive to yield a profitable return. This has long been a standard practice in cotton, an extravagant planting rate being used to avoid losses from seedling blight, and in the culture of canning tomatoes in curly top areas it has been found that setting two plants per hill in most cases allows one to escape even though the other is destroyed by the curly top virus.

Still another related phenomenon is seen in the escape from exclusively

insect-borne diseases by those varieties of plants that are repellent to the insect vectors. The plants may be susceptible but are not infected in nature since the vectors avoid them. A case in point concerns the "resistance" against mosaic of the raspberry variety "Lloyd George."

It is important to distinguish between true disease resistance and disease-escaping (klendusity), for a disease-escaping or klendusic plant may be destructively attacked by disease when grown under conditions that do not allow the escape function to operate.

Hypersensitivity. Hypersensitivity refers to the reaction of plants against obligate parasites in which the first cells attacked soon die, thus cutting off the pathogen from access to living cells and preventing its further development. It is best seen in the resistant reactions of plants toward rusts. A rust infection represents a delicately balanced equilibrium between pathogen and host in which the success of the infection depends on the host's having enough susceptibility to allow the fungus to nourish itself and reproduce, but not too much susceptibility, or the cells will die at once and with them the fungus. In breeding for disease resistance it is important to distinguish hypersensitivity and true resistance since a cross between a resistant parent and a hypersensitive parent (both appearing resistant although one is in reality excessively susceptible) might produce extremely susceptible offspring.

Acquired Immunity. Much of modern medicine is based on acquired immunity of animals. A patient recovers from scarlet fever; his blood contains antibodies that destroy the pathogen; he has acquired an immunity from scarlet fever such that he will not again develop the disease, and his blood serum may be used to prevent or cure scarlet fever in other patients. Once a child is inoculated or vaccinated with the virus of cowpox, a mild strain of the smallpox virus, he suffers a mild disease and, as a consequence, acquires immunity from the more virulent smallpox. Do plants have the same ability as animals to recover from one attack of disease and thereby acquire an immunity against subsequent attacks of the same disease? And can we make use of this in plant disease control?

Much thought has been given to these questions, but as yet there are few clearcut instances of acquired immunity in plants. The best evidence available concerns the virus diseases of plants as brought out in the case of tobacco ring spot.

Ring spot is a virus disease that results in severe necrosis of the leaves of young tobacco plants. If a diseased plant is protected and allowed to continue growth, the severe phase of the disease passes, the new leaves produced show less and less necrosis until finally leaves are produced that are entirely normal in appearance. If these leaves are again inoculated with

ring spot virus no further disease will develop: the plant has acquired immunity from ring spot (Fig. 158). Cuttings may be taken from the recovered parts of such plants and from these cuttings new plants may be produced. These appear entirely normal and they are immune, as they cannot again be made to show symptoms by inoculation of the virus. They still contain the virus for if juice from a "recovered" plant that appears normal is inoculated into a normal plant, the latter will develop typical ring spot. These recovered plants are like the notorious "Typhoid Mary" who recovered from typhoid fever, developed immunity thereby, yet continued for many years to pass on the disease to those with whom she came in contact. Moreover, as in animal medicine, the immunity is specific. The recovered ring spot plant is immune from ring spot but not from mosaic or any other disease, just as the patient who has recovered from or who has been vaccinated against smallpox is immune from that disease but not from any other type of human disease.

In some cases, then, plants recover from plant disease and display specific acquired immunity as a consequence. The next question concerns the uses to which this knowledge can be put.

Acquired immunity in plants has proved a useful tool in the difficult field of virus classification. Many viruses show variation and occur as distinct strains or races, each strain producing distinct symptoms on test plants. A hundred or more such strains have been detected in tobacco mosaic alone. In connection with quarantine laws, for example, it is important to know which viruses are strains of the same type virus and which are entirely distinct viruses. The recovered plant is immune from the various strains of the same type of virus but not from distinct but similar appearing viruses, and so the immunity test has come to be a valuable aid in determining the identity of viruses. By means of this test, for example, it has been shown that the viruses of celery mosaic and lily mosaic are both strains of cucumber mosaic virus, and that viruses of peach yellows and little peach are strains of the same virus.

So far little has been done in using acquired immunity for the practical control of plant diseases, although some work has been done along this line on potato viruses in England. The greatest possibilities concern plants that are vegetatively propagated, since in such plants a single "vaccinating" inoculation will be carried on to all vegetative progeny, or in plants that are extensively handled in culture, such as tobacco or tomato, in which the "vaccinating" inoculation of mechanically transmissible viruses could be automatically performed by having the operator keep his hands wet with attenuated virus juice during transplanting or some other handling operation. There is some danger in this method of disease control, how-

ever, since a widely distributed "vaccinating" virus might pass to another host where it could be destructive, might mutate to produce damaging strains, or might combine with a second mild virus to cause a harmful disease.

Complexity of the Factors That Determine Resistance. As a general rule, the defense mechanism of a plant, as that of an army, does not depend on any one structure, product, or function, but is a combination of many types of armaments. For a plant to be susceptible to a pathogen a long list of requirements must be fulfilled. In the average case of resistance, several of these requirements may be lacking. This is the reason for the difficulty in determining the exact nature of resistance in given cases, yet we must learn what we can of the nature of resistance to give us guidance in using this resistance in breeding for disease control.

Control of Plant Diseases through the Use of Resistant Varieties

The Need for This Type of Disease Control. In the long run control by resistant varieties is the best and cheapest method of disease control. In fact, there are many diseases that can be controlled in no other way, in particular those caused by persistent soil-dwelling fungi. Such diseases as watermelon wilt, flax wilt, tomato wilt, sugar cane mosaic, and curly top of sugar beets gravely threatened or even destroyed great agricultural enterprises before disease-resistant varieties of these crops became available, as no other effective control measures are known. Other crops are suffering important losses from diseases that cannot be controlled by any known means, and probably will not be controllable until resistant varieties have been developed.

Requirements for an Acceptable Resistant Variety. Fortunately many plant diseases are so host-specific that only certain varieties of a species or certain species of a genus are subject to a given pathogen. By crossing the resistant species or varieties with susceptible ones it is possible sometimes to obtain hybrids that combine the desired disease resistance of the one parent with desirable commercial and cultural characteristics of the other. It is not enough to secure disease resistance alone; many other characters must be combined with disease resistance before a variety is acceptable to growers and consumers. In Florida, for example, there is a project for breeding watermelons resistant to *Fusarium* wilt. While wilt resistance takes first place among the objectives of the work, consideration is given also to many other melon characteristics, including resistance to other diseases and insects, vigor, earliness, prolificacy, shape, size, color, rind thickness and texture, flesh color and texture, uniformity,

flavor, sugar content, seed size, color, and number, and market preferences. The melons that are sought in this project are those which will be substantially wilt-resistant but which must at the same time possess a majority of desirable characters of the types listed, for grower and market preferences are so strong that even in the face of heavy wilt losses it may be almost impossible to have a good resistant variety accepted by growers if it varies in any important degree from the type of susceptible melons which they have been growing.

Granted that a resistant variety is of an acceptable type, its resistance must meet rigorous requirements. It must hold up under widely varying growing conditions, and under conditions of heavy infestation. It must be genetically stable so that progeny of resistant parents also will be uniformly resistant. If more than one strain or race of the pathogen is prevalent the variety should carry resistance to all pathogenic strains to which it is likely to be exposed. In the face of these rigid requirements it is indeed astonishing to consider the accomplishments of breeding for disease resistance which have occurred within a period of less than 50 years.

Accomplishments in the Development of Disease-resistant Varieties. Chronologically, asparagus rust was one of the first plant diseases to be combated by means of resistant varieties. The disease appeared in previously healthy American fields in 1896 and within six years had spread from coast to coast. It was destructive everywhere and attempts to control it by dusting and spraying were unsuccessful. It was observed that some varieties were more subject to rust than others, and J. B. Norton of the U. S. Department of Agriculture imported numerous foreign asparagus varieties in a search for a truly rust-resistant one. Among these was one from England, "Reading Giant," which was most rust-resistant of all. It was variable in type but pure lines selected from hybrids of this and other varieties have given us such improved rust-resistant varieties as "Mary Washington" and "Martha Washington" which now dominate asparagus growing in America. Thus the problem of asparagus rust was solved within five years.

In 1895 the wilt disease of cotton was becoming alarmingly destructive in the area where Sea Island cotton was grown. A farmer, E. L. Rivers, encouraged and aided by Erwin F. Smith and W. A. Orton of the U. S. Department of Agriculture, began selecting the more resistant individuals in his fields and in two years had secured a tolerably wilt-resistant strain, but one with fiber which was of inferior quality. Better resistant strains were sought as it became evident that no other control measure would succeed. By 1902, Rivers had succeeded in isolating and multiplying a

good quality, wilt-resistant strain which was distributed as the new "Rivers" variety, while Orton had found another desirable resistant type which was soon widely cultivated as the "Centerville" variety. Meanwhile, the disease was becoming important in upland cotton, and Orton continued selecting from a partially wilt-resistant variety, "Jackson Limbless," eventually producing "Dillon," the first upland wilt-resistant cotton developed by systematic methods. Its other characters were unsatisfactory, however, so the search continued, with the result that "Dixie," a cotton of much better quality, was secured. Each of these had been produced by selecting a resistant individual, planting its progeny in a single row, and reselecting until the resistance became stabilized. Now the importance of hybridizing was recognized, and Dillon and Dixie were crossed with other desirable but susceptible varieties to produce a number of new resistant varieties, chief of which were "Dixie Triumph," "Dixie Cook," and "Cook 307-6." Continued selection and breeding has given us a number of high quality wilt-resistant varieties which are widely grown today.

In attempts to control the destructive wilt of watermelons, Orton crossed the watermelon with the resistant citron, producing the synthetic variety "Conqueror," a poor quality but wilt-resistant melon, the forerunner of a long list of desirable melons of various types.

It is not necessary to multiply instances of the efforts on many fronts to provide agriculture with more and better disease-resistant varieties of crops. Successful efforts have given us rust- and smut-resistant small grains, wilt-resistant tomatoes, alfalfa, flax, sweet corn, cabbage, and cowpeas, mosaic-resistant sugar cane, tobacco, and legumes, curly top-resistant sugar beets, blight-resistant potatoes, root rot-resistant sorghums and tobacco, nematode-resistant beans and cowpeas, mildew-resistant cucumbers and cantaloupes, and many others (see Frontispiece and Figs. 210 and 211).

Genetics of Disease Resistance in Plants. The earliest attempts at producing disease-resistant varieties consisted of selecting resistant individuals from susceptible populations, multiplying these individuals, and continuing reselection until pure resistant lines were obtained. Later and more effective efforts have been directed at deliberately producing resistant varieties by crossing resistant with susceptible parents, selecting from the hybrid generation, and backcrossing the selected individuals with the more desirable parent until desirable, homozygous, resistant lines have been secured. It has been seen that disease resistance is a genetic character as regularly inherited as any other genetic character, however the genetic behavior of resistance differs from one case to the next.

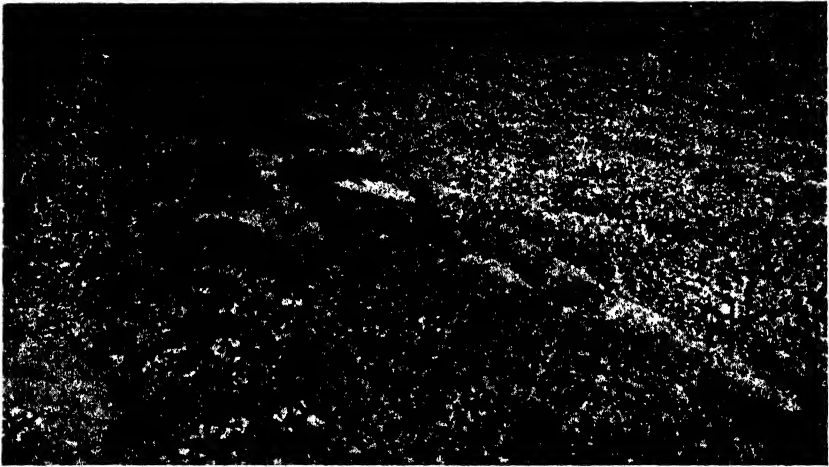


FIG. 211. Another of the noteworthy achievements in breeding for disease resistance was the production of powdery mildew resistant cantaloupe No. 45 by the U. S. Dep. of Agriculture. The resistant variety is seen in the two rows in the foreground bordered on both sides by rows of susceptible cantaloupes in which this destructive disease has largely defoliated the plants. (Photograph, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Dep. Agr.)

Resistance to one disease is not necessarily correlated with resistance to another. Thus Thatcher wheat, while resistant to stem rust, was so highly susceptible to leaf rust that it was replaced on that account; some of the wilt-resistant sweet corns are highly susceptible to smut, numerous wilt-resistant cottons are nematode-susceptible, and the "Victoria-type" of resistance to crown rust in oats is correlated with high susceptibility to a *Helminthosporium* disease. Nevertheless, by suitable breeding methods it is possible sometimes to combine the genes for resistance to several diseases in a single plant.

Simple monohybrid inheritance of resistance, depending on a single gene with a 3:1 Mendelian ratio, has been observed in numerous instances, as in the resistance of cabbage to yellows, of lettuce to downy mildew, and of peas to wilt. In these cases resistance behaves as a dominant character. Simple monohybrid inheritance of resistance with resistance recessive is seen in resistance to powdery mildew of barley, smut of sorghum, and speckled leaf blotch of wheat. Two-gene segregation (ratios 9:3:3:1, 15:1, 9:7, 3:13, or 12:3:1) appears to be involved in resistance to wilt and mosaic of beans, and is one type of inheritance in certain of the cereal rusts. Again, the resistant genes may be either dominant or recessive. Three-gene segregation (27:37) is seen in bean anthracnose, in a variety of beans which is resistant to three of the four races of the anthracnose fungus, resistance to each race being governed by a separate gene. Polymeric

genes (number unknown) for resistance also have been found as in the resistance of cucumbers to powdery mildew, of cotton to wilt, of tobacco to root rot, and of corn to scab and smut. Finally, instead of two allelomorphs for resistance or susceptibility there may be several allelomorphs giving a complex series of resistance relationships when several parents are studied.

In relation to physiologic specialization of the pathogen, a single gene for resistance may apply to only one race of the pathogen, as in the case of bean anthracnose, noted above, or a single gene may carry resistance to several pathogenic races, as in the resistance of wheat and oats to stem rust. Moreover, a given gene for resistance may function only at a certain period in the development of the plant or only under certain environmental conditions. This is well demonstrated in the cereals where rust resistance commonly applies to the mature plant, the seedlings of resistant varieties often being highly susceptible.

Limitations of Breeding for Resistance. Breeding for disease resistance may be impossible or only partly effective for a number of reasons:

1. **THERE MAY BE NO SOURCE OF RESISTANT GENES.** Breeding is usually most successful in dealing with a pathogen of narrow host range where some resistance is found within the genus or species of crop concerned. There are exceptions to this rule, however, as in the case of tobacco black root rot where success has been obtained in breeding root rot-resistant tobacco in spite of the wide host range of the pathogen. With a pathogen of very wide host range, such as that of Texas root rot, breeding for resistance may be unsuccessful because there is no starting point, no variety or species of cotton, alfalfa, or other hosts that manifests the least resistance to the pathogen. Miraculous as the work of the breeder appears to be, he must still have genes for resistance to start with.

2. **THE SOURCE OF RESISTANT GENES MAY BE TOO DISTANTLY RELATED TO THE CROP.** In bacterial blight of cotton a high degree of resistance is found in certain Asiatic cottons such as *Gossypium cernuum*, but these are so distantly related to American upland cotton that fertile hybrids between the two cannot be obtained. Intermediate cottons must be used and the genes for resistance must be passed slowly and step by step from the resistant species to the desirable but susceptible one. Increasing the number of chromosomes in a breeding parent, by colchicine or other treatments, helps in making some of these difficult crosses.

3. **IT MAY BE DIFFICULT TO COMBINE DISEASE RESISTANCE WITH THE OTHER DESIRABLE CHARACTERS.** At times the breeder encounters a genetic linkage between disease susceptibility and some other desired character so that he must take his choice between resistance and the other char-

acter; he cannot readily obtain both in the same plant. This has been a limitation in breeding smut-resistant corn since susceptibility and vigor are linked and vigor is essential in field corn. In early breeding of sugar beets for curly top resistance an undesirable tendency to bolting was associated with resistance, but this was later overcome.

4. IT MAY BE NECESSARY TO DEVELOP RESISTANCE AGAINST MANY PATHOGENIC RACES AT ONCE. It has been seen that the genes conditioning resistance to one pathogenic race or group of races may be distinct from those governing resistance to other races. To secure a variety that is practically resistant under field conditions it may be necessary to incorporate many genes into the variety until it is protected against all races to which it may be subjected. Remarkable accomplishments along this line have marked the progress of breeding rust- and smut-resistant cereals, but even here the unexpected appearance of a new or unrecognized race may nullify the breeders' efforts. Such races are appearing constantly, and this means that new resistant varieties must be developed constantly to replace old ones. If the breeder can keep a few years ahead of the disease that is all that can be expected; there is no time to sit back and rest on the laurels of an accomplishment in breeding for resistance. Even while a new resistant variety is being distributed, its successors must be in the early state of development.

5. INCREASED HOST RESISTANCE PROMOTES INCREASED VIRULENCE OF THE PATHOGEN. The development of a disease-resistant crop variety often is followed by its widespread cultivation. In cases such as that of powdery mildew of cantaloupe in California, corn wilt in Iowa, and tomato leaf mold in Ohio, the introduction and general adoption of disease-resistant varieties has been followed by apparent loss in resistance. On a resistant variety only old or new strains of the pathogen that are highly virulent can survive. The resistant variety filters the strains of low virulence out of the pathogen's population, leaving only the most highly virulent strains. This raises the general level of virulence of the pathogen, and accelerates the pace with which variation in the pathogen and natural selection of the most virulent races will ultimately overcome the resistance of the host.

6. THE PROBLEM OF CROSS FERTILIZATION. Once a resistant variety is developed it may be relatively easy to maintain its genetic purity if the plant is close-pollinated. But in dealing with a plant such as rye or sugar beets where cross pollination is the rule, every effort must be made to protect the new variety from genetic contamination, or the work of the breeder will be lost. "Meade" cotton, while it was not wilt-resistant, had other fine characteristics and commanded a premium price. By 1920-1922 it was planted on 10,000 acres and was becoming a striking success, but

mixing of seed, and planting close to other varieties so contaminated it that the fiber was rejected by the trade and by 1928 the variety was largely abandoned.

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Chapter 20

Control of Plant Diseases by Cultural Methods

Production of disease-free crops involves care and forethought at every step from the selection of the land on which the crop is to be grown until the final disposal of the harvested crop. It includes the planting of noninfested seed in noninfested soil (uninfested or disinfested), care and protection of the growing crop, and extends through the various steps of harvesting, storing, transporting, and marketing.

Noninfested Soil

Noninfested soil does not necessarily mean sterile soil nor soil that is free of all plant pathogens. It means soil that is relatively or entirely free of destructive pathogens capable of attacking the particular crop to be grown. Root knot nematode-infested soil may be looked upon as noninfested so far as grain crops and this pest are concerned. Noninfested soil may either be naturally free from dangerous pathogens (uninfested) or pathogen-free by virtue of treatments that have destroyed pathogens originally present (disinfested).

UNINFESTED SOIL

Soil can be considered uninfested when it is noninfested because of three conditions: it may be new land; it may be noninfested owing to the practice of rotation cropping; or it may be so owing to sanitation.

New Land. New land that has not been cultivated within recent times is often a desirable source of uninfested soil and in the past has been the only such source for crops affected by certain of the soil-borne wilt diseases (flax, banana). But even new land may be dangerous if, as sometimes happens, the wild native flora is harboring pathogens capable of attacking the cultivated crop. Thus, newly cleared oak land is a regular source of the fungus causing shoe-string root rot of orchard trees, and in the Southwest recently cleared land often contains the Texas root rot organism which is enphytotic as a benign parasite of the wild mesquite. Scab, *Fusarium* wilt, and *Rhizoctonia* of potatoes occur frequently in virgin soils planted with apparently healthy tubers. In general, grasslands may be

considered uninfested as regards diseases of dicotyledonous plants, although even in this case exceptions occur.

Rotation and Fallowing. In dealing with disease-producing organisms that live for a limited period of time on the debris of infected plants, land planted to nonsusceptible crops ordinarily purges itself of the organisms in shorter or longer periods—one to five years in many cases, although 10 to 20 years may in some cases be necessary. This riddance of such pathogens is due to their dying out in the absence of suitable host plants. As saprophytes they appear to be unable to maintain themselves in competition with the true saprophytes of the soil. Use of green manure crops in the rotation or application of organic matter in other ways aids in this self-purification of soil and is useful in controlling such soil-borne diseases as root knot, take-all of wheat, and Texas root rot.

In the case of diseases caused by organisms that commonly live as saprophytes even in virgin soil, rotations may sometimes be helpful. Here the length of time between susceptible crops is not the leading factor, but rather the types of intervening crops. This has been observed in potato culture in Nebraska where scab, wilt, and *Rhizoctonia* occur on virgin soils and in some rotations but are reduced to a minimum when alfalfa is introduced in the rotation just preceding a potato crop.

Rotation and fallowing are accessory control methods for many diseases, and in some cases give the only effective control of soil-borne pathogens. In addition, rotation and fallowing increase the fertility of soil, proving beneficial in the case of those diseases that are least destructive in well-nourished, vigorous host plants. To accomplish these purposes the period of persistence of the pathogen in the soil must be known, and for the rotation cycle to be fully effective it must be long enough to accomplish its purpose, the intervening crops must be nonsusceptible and it should preferably include soil-improving crops.

Sanitation. In connection with soil, sanitation includes:

1. **DISPOSAL OF INFESTED CROP RESIDUES** by burning, plowing, or feeding to swine or poultry. Recall that many foliage and fruit disease organisms persist as saprophytes in crop debris and sometimes complete their life cycles in this fashion.

2. **AVOIDING USE OF INFESTED MANURE.** This is an important source of soil infestation in the cases of corn smut, brown spot of corn, and potato scab. Some fungus spores are destroyed by passing through the digestive tract of farm animals, but even in such cases enough uneaten inoculum usually remains in the stall or pen litter to contaminate the manure.

3. **BARRIERS.** Barriers occasionally are effective in preventing the dissemination of soil-borne pathogens that spread mainly or only by growth

through the soil. Thus trench barriers sprinkled with creosote or oil, empty trenches, or living barriers of resistant crops are used in connection with such diseases as Texas root rot (Fig. 79), shoe-string root rot, and the nematode disease of sugar beets. To be effective, the barrier must be deep and wide enough to prevent spread below or across it and care should be taken not to carry inoculum across the barrier on shoes or tools. If the pathogen has an effective air-borne or insect-borne stage, barriers are unlikely to be of any value as control measures.

4. AVOIDING TRANSFER OF INOCULUM ON TOOLS. Contaminated soil on plows, wheels, hooves, and feet often is responsible for infestation of soil, as are contaminated mower blades and hand tools, particularly in the case of bacterial diseases. With bacterial ring rot of potato, contamination of healthy tubers by tools, containers, or contact with diseased seed pieces is the only means of spread. Wiping the tools with a disinfectant such as mercuric chloride, iodine, calcium hypochlorite, kerosene, or formaldehyde solution may be desirable to avoid this, and the grower should be aware of the danger in passing from infested fields to noninfested ones.

DISINFESTED SOIL

Disinfested soil is a practical means of securing pathogen-free soil when small amounts are concerned, as in greenhouses, seed beds, small ornamental plantings, and cold frames. In the past, cost of soil treatment usually has prevented its use on a field scale, but with the progressive development of cheaper soil disinfectants, larger and larger areas of valuable land can be treated economically. Complete sterilization of soil is neither necessary nor desirable and the emphasis today is on selective chemical disinfestation or soil pasteurization at temperatures that kill the more dangerous pathogens without detriment to the normal beneficial flora or physical properties of the soil. Although there are occasional cases of soil injury from disinfestation, in the majority of cases the treatment is beneficial as regards both disease and fertility. It hardly need be mentioned that soil disinfestation is useless if the treated soil is put into contaminated pots, flats, or beds, or if carelessness permits subsequent infestation of the soil.

Soil Disinfestation by Heat: 1. STEAM. Heat in some form usually is regarded as the best means of soil disinfestation, and, of the various means of applying heat, steam, either free or under pressure, is most widely used and most highly recommended. It may be applied in any of several ways:

BUILT-IN TILES OR PIPES a foot or more under the soil level, with frequent steam jets, represent the best means of disinfecting permanent beds, as in greenhouses and hotbeds (Fig. 212). While the original cost of



FIG. 212. Two methods of steam sterilization for greenhouses. (A) Installing a permanent tile system. While the initial cost is higher, the cost of operation is considerably less than in temporary installations, and the tiles can also be used for subirrigation and leaching. (B) The temporary buried-pipe method. Seven pipes, perforated with steam outlets, run the length of the bed. After steaming for a few hours or less under a canvas cover, the system is moved to a new location in the bed. (Photographs, Dep. Plant Pathology, Cornell Univ. Agr. Exp. Sta. & Extension Serv.)

installation is higher than with other methods, this extra cost is more than saved later on in labor and time economy. The system may also be used for subsoil irrigation.

STEAM PAN AND STEAM RAKE. In steaming soil in temporary locations or isolated infested spots, or where no built-in system is available, the inverted steam pan or steam rake may be used (Fig. 213A, B). The inverted steam pan is a square or rectangular galvanized iron pan measuring 6 ft. \times 6-9 ft. \times 8-9 in. deep or larger. This may be perforated in the bottom to receive a steam inlet pipe, or a flexible steam hose may be buried under the edge of the pan with the free end above the soil surface within the pan. Steam, supplied by a portable boiler or otherwise, enters under pressure and is allowed to flow for 20 to 60 minutes before the pan is moved on to the next location, slightly overlapping the former one. Similar results are obtained by covering greenhouse benches or beds with rubberized cloth or Sisalkraft paper and releasing the steam under this cover. A simple test of the time required for efficient disinfestation is to bury a medium-sized potato a few inches under the soil, and steam the soil until the potato is soft. The steam rake is a pipe with numerous fingerlike steam jets which are pressed into the soil, and allowed to deliver steam until a sufficient degree of disinfestation is obtained.

PRESSURE STERILIZERS. These are used for greenhouse soil in flats, for pots, and whenever highly efficient disinfestation of small lots of soil is required. A convenient type consists of a cement box about 30 in. \times 6 ft.



A



B



C

FIG. 213. Methods of soil disinfestation by steam. (A) Disinfesting field soil with an inverted pan serviced by a steam tractor. (B) A small inverted pan for greenhouse benches. (Photographs, Dep. Plant Pathology, Cornell Univ. Agr. Exp. Sta.) (C) Cement steam box with capacity of 36 standard flats for sterilizing potting soil, pots, and infested greenhouse debris.

× 30 in. deep with side walls 4–6 in. thick, with steam inlet from the greenhouse service line and a tightly fitting wooden cover. Flats of soil or pots are stacked in it with circulation spaces between, and steamed for 1 hour or more at about 5 lbs. pressure (Fig. 213C).

2. DRY HEAT. This must be used at higher temperatures than steam for equivalent disinfestation.

FIRING by building a fire over the soil to be treated is perhaps the

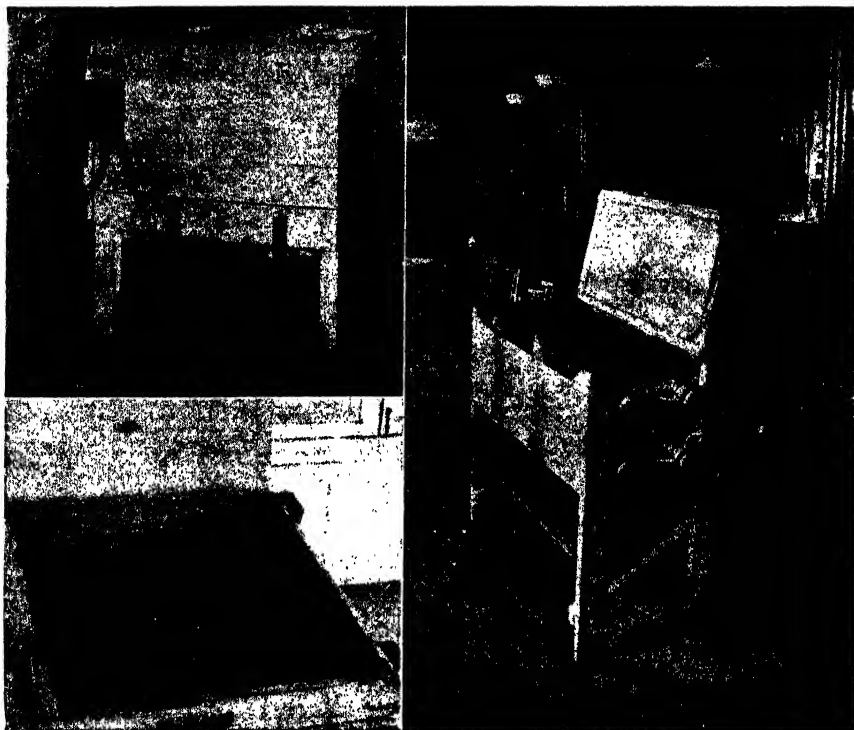


FIG. 214. Three types of equipment used in electric pasteurization of soil. (Top, left) A pasteurizer of $\frac{1}{2}$ cu. yd. capacity that will treat 11 bushels of soil in 4 to 5 hours. (Right) A homemade 4-bushel electric pasteurizer of indirect-heating type. (Bottom, left) A portable 2-bushel sterilizer that treats a charge of soil in 4 hrs. Note the heating coil. (Photographs, Dep. Plant Pathology, Cornell Univ. Agr. Exp. Sta.)

oldest known method of soil disinfestation, but is too drastic and uncertain for ordinary use. It is used to some extent in sterilizing tobacco plant beds.

BAKING in ovens is an improvement over firing but unless carefully done is likely to injure the soil.

DRY HEAT PASTEURIZATION at controlled temperatures is a recent and valuable development. Various types of home-made or custom-built electric ovens may be used (Fig. 214). Among the modifications are an electric inverted-pan pasteurizer operating on the same principle as the inverted steam pan, and a continuous-flow electric soil pasteurizer operating on the same principle as a baker's oven. In any case, soil thus heated to 160°F. is considered to be adequately pasteurized.

GREENHOUSE OVERHEATING. The following method of dry heat disinfestation was reported by a grower as highly effective in nematode control and appears to be worthy of consideration. In August, when the green-

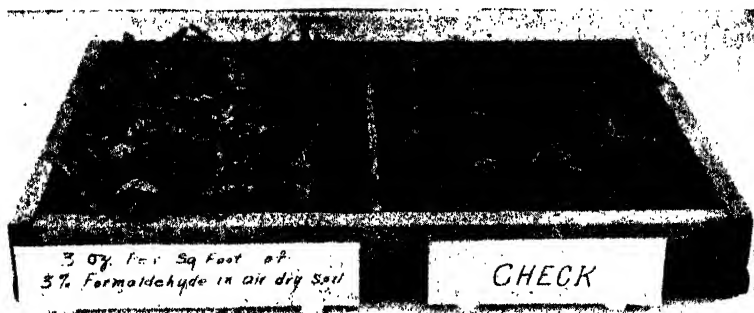


FIG. 215. The use of formaldehyde dust in preventing damping-off of seedlings. (Left to right) The seedlings are calendula, clarkia, zinnia, and kochia. (Courtesy, P. E. Tilford, Ohio Agr. Exp. Sta.)

house contains no plants, all flats, pots, and soil to be disinfested are placed in a greenhouse on a hot day (100°F. or higher), and the heating system is turned on at full capacity. Within a short time such high temperatures are reached that the greenhouse and all its contents become thoroughly pasteurized.

Soil Fumigation. Chemical soil fumigation has several purposes: to destroy pathogenic fungi and bacteria, insects, nematodes, and weed seeds. A great variety of chemicals have been used for this purpose, most of which are effective against one or more of these pests but not against all. In undertaking soil fumigation it is important to consider the object of the treatment, the cost of the chemical in relation to the value of the treatment, possible salvage of some of this cost through reduced weeding and fertilizer value of the chemical, changes resulting in the soil properties, possible harmful chemical accumulations in the soil, and safety hazard in application.

1. **FORMALDEHYDE.** In the past, formaldehyde has been in common use for soil fumigation. The usual dosage is a 1:20 to 1:50 dilution of commercial formalin applied at the rate of 2 qts. per sq. ft. of soil surface. The soil is spaded up, covered with a tarpaulin or wet sacks for 24 hours, then aerated until the odor has disappeared before using. At present formaldehyde dust is to some extent replacing the liquid as no aerating period is required before planting. The dust consists of formalin adsorbed to an inert carrier. A good mixture is 15 parts formalin plus 85 parts fine charcoal or humus, applied at the rate of $1\frac{1}{2}$ oz. per sq. ft. of soil. It is mixed with soil, planted, and then watered (Figs. 126 and 215). Another practice that is useful for small lots of greenhouse soil is to mix one part of formalin with 5 to 10 parts of water, then sprinkle the solution on the soil, which is planted and watered at once. Very dilute formaldehyde may be sprinkled

Table 8
DATA ON SOME STANDARD AND POTENTIAL SOIL NEMACIDES*

Fumigant Chemical	Trade Name	Dosage rates†		Cost of materials‡		Effective controls§			Persistence in soil
		Per 100 sq. in. (cc.)	Per 1000 sq. ft. (lbs.)	Per lb.	Per 1000 sq. ft.	Nemas	Fungi	Weeds	
Carbon disulfide.....	..	6.0	24.1	\$.08	\$1.92	Good	Variable	Fair	Short
Chloropicrin.....	Larvacide	2.0	10.5	.80	8.40	Good	Good	Fair	Medium to long
Dichloropropylene + dichloropropane.....	DD, Dow- fume-N	1.9	7.0	.15	1.00	Good	Poor	Poor	Long
Dichlorisopropyl ether.....	..	2.0	7.0	.15	1.05	Good	Long
Methyl bromide.....	..	0.6	3.3	.65	2.15	Good	Fair	Fair	Very short
Methyl bromide + ethylene dichloride + carbon tetrachloride.....	Dowfume-G	6.0	26.3	.20	5.26	Good	Fair	Fair	Very short
Methyl bromide + xylene.....	Isobrome	4.0	12.6	.35	4.42	Good	Fair	Fair	Very short
Ethylene dibromide.....	Dowfume- W40, Isco- brome-D	1.0	3.2	.26	.92	Good	Poor	Fair	Long
Ethylene dichloride.....	..	10.0	40.0	.08	3.20	Good	Poor	Poor	Short
Pentachlorethane.....	..	9.6	50.0	.20	10.00	Good	Poor	Poor	Long
Tetrachlorethane.....	..	10.0	44.7	.19	8.49	Good	Poor	Good	Very long
Xylene.....	..	8.0	21.6	.05	1.08	Good	Good	..	Medium

* Condensed and amended from *Soil Science*, 61, 77 (1946); published with permission of The Williams and Wilkins Co.

† Dosages given are currently believed to be satisfactory for commercial root knot control in sandy soil at 70°F. when injected in 10-in. centers and sealed in with water. A few of these materials probably do not need to be sealed in. The dosage for ethylene dichloride may be too low.

‡ Not all prices are authorized and usually represent lowest available price in drum lots.

§ Much variability in effectiveness may be expected under different conditions and for different pathogens; therefore, ratings are tentative at best.

|| Usually a long period of persistence in the soil means considerable toxicity to subsequent plant growth, whereas a short period of a week or less denotes relatively little toxicity.

on beds immediately after planting. Still another method of soil disinfection that is highly effective is use of a combination of formaldehyde and steam.

Formaldehyde leaves no harmful or useful residue in the soil. It is fairly effective against some pathogens, but only incompletely so against root knot nematodes. The use of a formaldehyde drench on a field scale is limited by the great amount of water required and the expense which amounts to \$300 to \$600 an acre.

2. ORGANIC FUMIGANTS. Recently a number of new, useful soil fumigants have been developed. Data on these are given in Table 8. None of these meets all the requirements of an ideal soil fumigant, each possessing both advantages and disadvantages. With improvements in methods of application and production, the expense and effectiveness of these may be expected to change. With gradual reduction in their cost, larger and larger areas of land can be treated economically.

For treating nursery, flower, and greenhouse beds and other small areas, the fumigant can be applied with a hand-operated soil injector of about one gallon capacity, while for larger tracts, horse- or tractor-drawn injectors are available. The gas is sealed in by watering the soil after treatment or by covering it with gas-tight paper (Fig. 216) except in large tracts where this is impractical.

To simplify soil fumigation for gardens or other small areas, where injection equipment is not available, dilute ethylene dibromide (Garden Dowfume) is available. This is simply dripped into furrows in the soil, and the land is then leveled.

Soil fumigants must be used with precautions. Some are irritating or



FIG. 216. Applying a soil fumigant to nursery soil for destroying root knot nematodes. The treated soil is covered with gas-tight paper, held in place by a border of soil. Space intervals of application are guided by a knotted string.

poisonous to breathe or they burn skin, clothing, or metal equipment. Some are inflammable. The fumigants are injurious or deadly to nearby vegetation and therefore cannot be safely used close to trees or shrubs. Treated soil cannot be safely planted until the vapors of the fumigant have become dissipated. This may take a week or longer, depending on the chemical, soil type, and temperature, and may be hastened by working the soil.

3. SULFURIC ACID and other acids frequently are used as soil treatments for reducing losses from damping-off in nursery beds. There is a question whether this is a *bona-fide* case of soil disinfection, or whether the beneficial effect is due largely to a shifting of the pH to a range in which the pathogens are inhibited.

4. CYANAMIDE, SODIUM CYANIDE, AND CALCIUM CYANIDE have been used for disinfecting field soils of the root knot nematode. With the cyanides ammonium sulfate is added to release hydrocyanic acid gas. While the cost is high (about \$100.00 per acre for materials) these materials have value as fertilizer.

5. MERCURY COMPOUNDS, both inorganic as the mercuric and mercurous chlorides and in proprietary organic preparations such as "Special Semesan" and "Nu-Green," are used sometimes as soil fungicides, often with very good results in preventing damping-off and lawn diseases caused by soil-borne organisms. It should be noted also that when fungicidal dusts are used for seed treatments a part of their effectiveness relates to their local soil disinfection in the vicinity of the sprouting seed.

6. COPPER COMPOUNDS, such as Bordeaux mixture and red and yellow copper oxides have an effect similar to that of the mercury compounds both when applied directly to the soil or when acting from seed surfaces.

7. NONMETALLIC ORGANIC FUNGICIDES. Some of these, such as Tersan (tetramethyl thiuramdisulfide), are used in a fashion similar to the mercury compounds for lawn diseases.

Noninfested Seed

In many cases internally or externally infested seed may be the means of introducing disease into a crop, even though the soil is free of dangerous pathogens. The list of diseases in which seed are the principal or only means of such introduction is a long one and includes such important diseases as the smuts of small grains, virus diseases of potatoes and legumes, bacterial diseases of legumes, cotton anthracnose, and numerous others. As in the case of soil, pathogen-free seed may either be naturally so, or rendered innocuous by physical or chemical treatments.

UNINFESTED SEED

Seed from Uninfested Areas. When a disease does not occur in a given part of the world, that area may be a useful source of disease-free seed. Use is made of this principle in a commercial way in the United States. Beans grown in Idaho usually are free from the pathogens of bacterial blight and anthracnose, and cabbage seed from the Puget Sound region does not carry black rot, in each case because the disease in question does not occur normally in those areas, rendering them valuable seed sources for the infested parts of the country.

Seed from Protected Seed Blocks. If seed for home use, even in a region of infestation, is grown in an isolated seed block, under rigorous conditions of care, pathogen-free seed may be harvested. The relatively small size of such blocks makes it practical to follow a control program scrupulously, even resorting to such practices as roguing, spraying, or dusting crops that normally are not so treated. Production of uninfested seed in special seed blocks is a standard practice, for example, in producing seed wheat free from the loose smut pathogen, or virus-free potatoes.

Indexed Seed. Indexing is used in the production of virus-free seed potatoes. The method is described on p. 333.

Cleaned or Selected Seed. While it is always better to use originally clean seed than to separate uninfested seed from an infested lot, selecting or cleaning seed has its value, not only as regards disease but also in order to avoid other seed-transmitted defects. Cleaning seed wheat by fanning to blow out the light smut balls is a necessary preliminary to seed treatment in the case of bunt, and is useful in control of scab and other wheat diseases. Pathogens which, strictly speaking, are not seed-borne may be carried in the fragments of hay in poorly cleaned seed but may be removed by fanning. Cleaning by flotation after delinting is a standard practice in removing infected seed from cotton and in separating ergot sclerotia or nematode galls from rye and wheat seed. Dodder seed are removed from alfalfa or clover by special machines used by the larger seed producers. Tubers infected with bacterial ring rot may be removed from seed potatoes by hand culling in a dark room under ultraviolet light, and hand selection to reject any obviously defective or infected propagating material is a standard practice with nursery stock, bulbs, corms, and tubers.

Certified Seed. Certified seed is commercial seed grown by reliable persons according to the rules for eligibility to certification for each crop in each state. Usually it carries an official blue label and is sold at a price up

to twice that asked for ordinary seed. It is the first or second generation crop from registered seed or foundation stock, which ordinarily is produced under the direct supervision of the state agricultural experiment station. The rules for certification vary somewhat from one state to another, but in all cases, in addition to demanding varietal purity and freedom from noxious weed seed and other undesirable contaminations, the rules permit only very small amounts of disease or in cases of certain serious diseases, none at all. Fields for certification are inspected once or twice, and the harvested seed also undergoes a bin inspection. The additional cost of certified seed is well justified in nearly all cases, particularly in dealing with diseases that cannot be detected in seed, but only in the field, such as loose smut of wheat, virus diseases of potatoes, seed-borne bacterial diseases, and certain mosaic diseases of legumes. Data from Wisconsin show that certified seed potatoes have increased yields in that state by 36 per cent on the average and up to 155 per cent in extreme cases. An extra cost of \$26.40 for certified seed returned a gain of \$68.40 and a net profit of \$42.00 per acre.

Registered Propagation Wood. As a rule, virus diseases of trees are disseminated by the use of scions or buds from infected trees, often without being suspected since the symptoms of these diseases frequently are not apparent at certain times when the wood is infective. To avoid this danger in connection with psorosis, a virus disease of citrus trees, the California Department of Agriculture maintains an inspection service of bud-parent trees, and those known to be free of the disease are registered, numbered, and tagged, affording citrus growers a source of high-grade propagative wood by a process comparable to the use of registered sires in livestock breeding. In several states there are similar programs of certification of stone fruit budwood, for virus control. On a less efficient scale the state nursery inspection services offer comparable aid to growers.

DISINFESTED SEED

The dual purpose of seed disinfestation is to kill disease inoculum inside or on the surface of seed and to kill or repel disease organisms in the soil about the seed. There are several requirements for a good seed disinfestant and few chemicals are not deficient in one or more of these. A good seed treatment must be effective in controlling the pathogens against which it is directed; it should be relatively simple, for if too complicated it will not be generally accepted; it should be practical, as in the case of hot water treatments of grain for small seed lots but not for the seed for large acreages; it should be inexpensive; and it should not be injurious to the seed.

Disinfestation by Chemicals. Disinfestant chemicals are applied to seed in various ways. In the older dip method, which is still widely used for potatoes and occasionally for vegetable and flower seed, the seed is enclosed in loosely woven sacks and immersed in the chemical solution, for a moment in the case of chemicals which are to remain on the seed, and for a stated length of time with chemicals such as formaldehyde, which evaporates, or corrosive sublimate which must be washed off the seed with water after the treatment. In the spray treatment, used principally with formaldehyde for smut control in oats, the chemical is sprayed or sprinkled over the seed as it is shoveled about (Fig. 21) or as it leaves the grain spout. The dusts are applied to small lots of seed by shaking the seed and dust in a closed jar or tin. With larger amounts of seed, the dust is applied in a home-made treater of the rotary barrel or gravity type (see Fig. 19) or in a power-driven commercial treater.

Two new methods of seed treatment are pelleting and the slurry method. In pelleting, the seed is covered with a quick-drying plastic, such as methyl cellulose (Methocel), in which is incorporated the seed disinfestant. This permits increasing the dosage of seed protectant beyond that which will adhere to uncoated seed. With the slurry method the seed is mixed with a small amount of a thick water suspension of disinfestant. The small amount of water quickly evaporates so that treated seed may be stored immediately. Advantages of the method are the elimination of the hazard of flying chemical dust and more accurate dosages. With corn, using a power-driven treater, up to 300 bushels per hour can be slurry-treated. Field performance compares favorably with that of dusted seed.

The principal chemicals used for seed treatment are listed in Table 9. The evolution of seed disinfestants has shown a change from inorganic oxides and salts of heavy metals (mercuric chloride, copper sulfate and carbonate, zinc and copper oxides) and formaldehyde to the organo-mercury dusts, and now to organic, nonmetallic compounds such as Spergon, Phygon, Arasan, and Fermate. The latter have the advantage of being much less hazardous to use, but lack the volatility of some of the organomercurials which still retain their place where the seed structure is such as to require a volatile disinfestant (cotton, oats, barley, broom corn).

Mercuric chloride and formaldehyde still have an important place in treatment of potato tubers (see pp. 92 and 296), and the former for tomato seed (see p. 275) and certain other vegetable and flower seeds and bulbs. The dosages of seed disinfestants, with methods and precautions for their use, usually are stated on containers and should be followed carefully.

Table 9

PRINCIPAL CHEMICALS USED IN PLANT DISEASE CONTROL

<i>Chemical</i>	<i>Common or Trade Names</i>	<i>Principal or Prospective Uses and Notes</i>
SULFUR AND ITS INORGANIC COMPOUNDS		
Elemental sulfur	Flowers of S, S flour, dusting S, inoculated S, wettable S, flotation S, micronized S, etc., under many trade names.	Sprays and dusts for apple leaf and fruit diseases, brown rot of stone fruits, cherry leaf spot, rose leaf diseases, powdery mildews, rusts, peanut leaf spot, iris leaf spot, soybean downy mildew; to acidify soil for potato scab, Southern wilt; as vapor for fumigating greenhouses, storage houses.
Lime-sulfur	Self-boiled lime-S, dry lime S, dry-mix lime S, etc., under many trade names.	Dormant or growing-season sprays for apple leaf and fruit diseases, brown rot of stone fruits, cherry leaf spot, elm leaf spot, sycamore anthracnose, cane fruit anthracnose, peach leaf curl, black knot of plum.
Sulfuric acid		Delinting cotton seed; soil treatment for damping-off in nursery beds and combating diseases favored by alkaline soil.
Carbon disulfide		Soil fumigant; nemacide. See Table 8.
INORGANIC HALIDES		
Carbon tetrachloride		Soil fumigant; nemacide. See Table 8.
Calcium hypochlorite Sodium hypochlorite	BK, Chlorox, etc.	Laboratory seed and tissue disinfectant.
Copper oxide	Red and yellow copper oxides, Cuprocide, Metrox, etc.	Seed treatment dust for sugar beets, vegetables and flowers except cruciferous plants; spray for post-emergence damping-off, rose leaf diseases, celery blights, tobacco and hops downy mildews.
Copper carbonate	Coppercarb, Carbo, and other brands	Formerly used extensively for wheat seed treatment, now largely limited to use on sorghum seed.

Table 9—(Continued)

<i>Chemical</i>	<i>Common or Trade Names</i>	<i>Principal or Prospective Uses and Notes</i>
Copper sulfate	Many brands	Ingredient of Bordeaux mixture and used alone as a spray for disinfecting storage houses. Eradicant spray for powdery mildews.
Basic copper chloride	Copper A	Spray or dust for beet leaf spot, cucurbit anthracnose, rose leaf diseases, celery blights, potato and tomato foliage diseases, apple fire blight, cucumber downy mildew, grape black rot, cherry leaf spot.
Copper oxychloride sulfate	COCS	
Tribasic salt of copper sulfate	Tribasic	
Copper phosphate and numerous other Cu salts	Insoluble or fixed coppers, copper-lime	
INORGANIC ZINC COMPOUNDS		
Zinc oxide	Vasco-4	Seed treatment for vegetables, except peas, and flowers.
Zinc sulfate-lime	Zinc-lime	Spray for apple leaf and fruit diseases, brown rot and bacterial spot of stone fruits, pecan rosette. Used with "Dithane" (see below).
Zinc chloride		Wood preservative
INORGANIC MERCURY COMPOUNDS		
Mercuric chloride	Corrosive sublimate, bi-chloride of mercury	Seed treatment dip for tomatoes, cucurbits, beans, Irish and sweet potatoes; tool and laboratory disinfectant.
Mercurous chloride	Calomel, Calo-Chlor	Spray or dust for turf diseases, alone or with HgCl ₂ . Soil treatment for club root of crucifers.
ORGANOMERCURY COMPOUNDS		
Ethylmercuric phosphate	New Improved Ceresan	Seed treatment for cereals, cotton, sugar beets, peas, flax, bulbs.
	Semesan Jr.	Seed treatment for corn, flower bulbs.
Ethylmercuric chloride	2 per cent Ceresan	Seed and bulb treatment for cotton, peas, flower bulbs.
Ethylmercuric- <i>p</i> -toluene sulfanilide	DuBay 1451, 1452, 1452F, Ceresan-M, GGG(slurry)	Replacing Ceresan for the same uses. Less toxic than Ceresan, but fully as effective.

Table 9—(Continued)

<i>Chemical</i>	<i>Common or Trade Names</i>	<i>Principal or Prospective Uses and Notes</i>
Hydroxymercuri chloro-phenol	Semesan	Seed treatment for vegetable and flower seed and bulbs.
Hydroxymercuri chloro-phenol + hydroxymercuri nitrophenol	Semesan Bel	Treatment for Irish and sweet potato tubers, roots, and sprouts, woody cuttings, and grafts.
Mercuric phenyl cyanamide + cadmium oxide	Barbak C	Seed treatment for corn.
Phenylmercuri triethanol ammonium lactate	Puratized N5 Puraturf	Spray for apple leaf and fruit diseases, bean anthracnose, cucurbit anthracnose; dip for Irish and sweet potatoes; for mildew-proofing textiles.
Phenylmercuri-2,2,2-nitrilo-triethanol	Puratized ND-5	Prevention of citrus fruit decay.
Phenylmercuric nitrate		In antiseptic tree paint to check sycamore canker.
Phenylmercuric acetate	Mersolite	Cottonseed treatment; mildew-proofing textiles.
Pyridylmercuric chloride, stearate, acetate		Mildew-proofing textiles, cork, leather, etc.
Sodium ethyl mercurithio-salicylate	Merthiolate	Prevention of citrus fruit decay
HALOGENATED ORGANIC COMPOUNDS		
Zinc trichlorophenate	Dow 9 A and B	Seed treatment for cotton, peas, and other crops; bulb dip.
Paradichlorobenzene	PDB	Fumigation for tobacco downy mildew control.
Pentachlorophenol		Wood preservative for fence posts, etc.
Methyl bromide, alone and in mixtures	Dowfume G, Iscobrome	Soil fumigant; nemacide. See Table 8.
Ethylene dibromide	Dowfume W10, W40, Iscobrome D	" " " "
Ethylene dichloride		" " " "
Pentachlorethane		" " " "
Tetrachlorethane		" " " "
Dichlorisopropyl ether		" " " "
Dichloropropylene + dichloropropane	DD, Dowfume N	" " " "
Chloropicrin	Larvacide	" " " "
Lauryl isoquinolineum bromide	Isothan Q15	Possible value as apple and cherry spray.

Table 9—(Continued)

<i>Chemical</i>	<i>Common or Trade Names</i>	<i>Principal or Prospective Uses and Notes</i>
Lauryl pyridinium bromide	Isothan Q4	Promising as spray for cherry leaf spot.
Dimethyl dilauryl ammonium chloride	Isothan Q-DLI	Spray for brown spot and copper spot of turf. (Other related compounds are under trial.)
CARBONYL COMPOUNDS		
Formaldehyde	Formalin, many brands	Soil fumigant; tool disinfectant; seed, tuber, and bulb treatment for potatoes, oats, flowers; disinfectant for storage houses.
Tetrachloro- <i>p</i> -benzoquinone	Spergon	Seed treatment for vegetables, flowers, cereals except oats and barley, field legumes; bulb treatment for lily, onion; root and sprout treatment for sweet potato; cutting treatment; spray or dust for cabbage downy mildew.
2,3-dichloro-1,4-naphthoquinone	Phygon	Spray for apple leaf and fruit diseases, cherry leaf spot, potato late blight, raspberry spur blight, tomato <i>Septoria</i> and anthracnose; prestorage dip for carrots; seed treatment for cereals (except oats and barley), peanuts, flax, some vegetables; root treatment for sweet potatoes.
CARBOXYLIC ACIDS AND DERIVATIVES		
Acetic acid		Tomato seed treatment.
Bismuth subsalicylate		Spray for tobacco downy mildew, tomato early blight.
QUATERNARY AMMONIUM COMPOUNDS		
Phenylmercuritriethanol ammonium lactate	Puratized N5	Listed under organomercury compounds. Related compounds are commercial bactericides with fungistatic properties.
Lauryl pyridinium bromide, etc.	Isothan Q4, etc.	This and related compounds are listed under halogenated organic compounds.

Table 9—(Continued)

<i>Chemical</i>	<i>Common or Trade Names</i>	<i>Principal or Prospective Uses and Notes</i>
THIOCARBAMATES		
Tetramethylthiuram disulfide	Arasan, Tersan	Seed treatment for cereals (except oats and barley), peanuts, vegetables, flowers; lily bulb treatment; treatment for cuttings of rose, carnation, sweet potato sprouts; mixed with fertilizer for onion smut control; spray for apple Brooks spot, tomato early blight, turf diseases; repellent insecticide.
Ferric dimethyl dithiocarbamate, with or without sulfur	Fermate, Karbam (black), Thionate Dust, du Pont Rose Dust	Spray or dust for major leaf, stem, and fruit diseases of apple, bean, cane fruits, carnation, celery, cherry, cranberry, cucumber, eggplant, elm, grape, peach, pear, pecan, rose, tobacco, tomato (anthracnose), tulip, walnut; seed treatment for vegetables, sorghum; root and sprout treatment for sweet potato; gladiolus bulb treatment; treatment for woody cuttings; as seed treatment or mixed with fertilizer for onion smut; spray for post-emergence damping-off; repellent insecticide.
Zinc dimethyl dithiocarbamate	Methasan, Zerlate, Zincate, Zimate, Karbam (white)	Spray for apple bitter rot and rust, carrot and celery blights, cranberry fruit rots, pear scab, potato early blight and leaf hoppers, tomato early blight and anthracnose, tulip gray mold, turf diseases.
Disodium ethylene bisdithiocarbamate, or this + ZnSO_4 + lime \rightarrow zinc ethylene bisdithiocarbamate	Dithane, IN5446, He-178	Spray for leading leaf, stem, and fruit diseases of apple, bean, cabbage, carnation, celery, cherry, cucumber, peach, pear, potato, raspberry, rose, tomato, walnut; reduces potato flea beetles, aphids, leafhoppers, Mexican bean beetles.

Table 9—(Continued)

<i>Chemical</i>	<i>Common or Trade Names</i>	<i>Principal or Prospective Uses and Notes</i>
OTHER ORGANIC NITROGEN COMPOUNDS		
Sodium dinitro- <i>o</i> -cresoxide	Elgetol, Sinox	Eradicant dormant or delayed dormant spray for apple scab, grape black rot, peach bacterial spot, leaf curl, pear blotch, leaf diseases, raspberry spur blight, thread blight of tung, turf diseases, etc.; for therapeutic treatment of cedar rust galls; and crown gall; wood preservative; insecticide; selective weed killer.
Dinitro- <i>o</i> -cyclohexylphenol	Dinitro, DN, DNOCHP	Uses similar to Elgetol; for bark shelling of citrus in psorosis control.
Copper 8-hydroxyquinoline	Copper 8	Standard bacteriostatic, used as chemotherapeutic for vascular diseases.
2-heptadecyl imidazoline (glyoxalidine)	Compound 341	Spray for cherry leaf spot.
MISCELLANEOUS AROMATIC COMPOUNDS		
Benzene		Fumigant for tobacco and cabbage downy mildews.
Naphthalene		Fumigant for tobacco downy mildew.
Diphenyl		Mild fungicide used in fruit wraps.

Disinfection by Heat: 1. HOT WATER SEED TREATMENTS for controlling internal seed infections are used in the following cases:

Loose smut and nematode disease of wheat (129°F., 10 min.)

Brown loose smut of barley (126°F., 13 min.)

Stem nematodes in ornamental bulbs.

Celery late blight (118°F., 18 min.)

Cabbage blackleg (122°F., 30 min.)

Tomato bacterial canker (122°F., 25 min.)

The use of hot water disinfection of propagating wood of virus-diseased stone fruits is discussed below.

2. DRY HEAT SEED TREATMENTS have been used in a primitive fashion in hot climates for preventing cereal smuts, by exposing the seed in

direct sunshine on very hot days. Dry heat seed (or tuber) treatments are rarely used today, but have proved effective in the following cases:

Bacterial wilt of corn (pasteurizing at 140°–158°F. for 1 hour).

Late blight of potatoes (104°–120°F. for 4 hours).

Cotton anthracnose (122°F., 36 hours; or 140°F., 24 hours followed by 205°F. for 8 hours).

3. VAPOR-HEAT SEED TREATMENTS, using moist heat from a vapor-heat machine, have the advantages of less critical temperature requirement to disinfect seed without injury to them, great volume of seed that can be treated at once, rapid removal of excess moisture from the seed, and application of the method on the belt conveyors used in commercial seed handling. The method is in the developmental stage.

Disinfestation by Aging. Some pathogens die out of seed before it loses its viability. This is true of the cotton anthracnose fungus, which can be removed from infested seed by aging for two years, although the viability of the seed is unimpaired. The pathogen of squash foot rot soon dies out of seed, and this disease now is controlled largely by standard use of two-year-old seed.

Care of the Crop

Choice of Site. As pointed out above, some locations must be excluded at the outset for cultivation of certain crops because of soil infestation. A striking example is the avoidance of land infested with root knot nematodes for nursery stock, or the production of root knot-susceptible vegetables or fruits. Where there is a choice of sites, other pathologic factors may decide which will be best, such as good air drainage for small grain culture or an apple orchard site that will permit the most efficient spraying or be most distantly removed from alternate hosts of the cedar-apple rust fungus. Potential disease problems as well as present ones should influence the choice of site.

Preparing the Soil. In many cases, prompt plowing soon after the harvesting of a crop is beneficial from the disease standpoint, to facilitate decay of the crop residue on which many pathogens are dependent, and to bury spores. This cannot be done in all cases, for other than pathologic reasons, e.g., in areas subject to wind erosion.

Soil Amendment. In some instances, soil amendment is a large element in disease control, as in the dry-land foot rot of wheat, in which weathered or undernourished plants are most subject to disease. Specific soil amendment practices are indicated in the case of cotton wilt (potassium), potato scab (sulfur), club root of crucifers (lime), bacterial spot of stone fruits (nitrogen), and all mineral deficiency diseases. In those diseases favored by succulence in the host (e.g., rusts and many bacterial

diseases) excessive nitrogen fertilization predisposes the plants to disease, and this can be avoided by preventing such fertilization and by applications of potassium and phosphorus to offset the nitrogen.

Seeding: TIME OF SEEDING. Time of seeding has an important bearing on disease control in many cases. Dry-land foot rot of wheat may be practically controlled by proper date of seeding. Early spring seeding may be effective in dealing with diseases such as root knot or Texas root rot which prevail only in the hot summer months. The effect of time of seeding usually is directly related to the influence of temperature on disease, as is strikingly seen in the case of bunt (inhibited by soil temperatures above 70°F.).

RATE OF SEEDING. Those fungus diseases that are favored by excessive humidity develop most destructively in the moist conditions accompanying excessive rates of seeding (cereal rusts and powdery mildew; damping-off), but where heavy seedling loss from disease characterizes a crop, as in cotton, such losses may be averted by excessive seeding rates to compensate for seedling mortality.

DEPTH OF SEEDING. Deep planting often favors damping-off and other soil-borne fungus diseases by lengthening the susceptible seedling stage. In addition, it may lead to root asphyxiation as also in the burying of plants with soil deposited by wind or water.

Water Supply. Where the water supply is controllable, it is often a leading factor in disease control. Excessive watering favors those pathogens that are dependent on moist air and soil, and in the case of bacterial diseases often aids in the spread of the pathogen. On the other hand, there are occasional cases where flooding serves as a disease control measure, as in the case of root knot, and brown spot and blast of rice. High-pressure water spraying sometimes aids in the control of diseases caused by superficial organisms, such as powdery mildew. An unusual case is that of cranberry leaf drop, a disease due to asphyxiation of the plants under water, which is controlled by flooding the beds in winter, allowing a crust of ice to form, then draining away the water beneath the ice to permit aeration under the ice crust. So important is water in relation to plant disease that the entire pathology of a crop may be profoundly altered under irrigation and in planning irrigation projects this potential hazard should be seriously considered.

Handling. In crops such as tobacco, tomatoes, and fruit trees requiring frequent handling (transplanting, grafting, pruning, staking, dis-budding, hand-pollination, etc.) the act of handling in itself is often a means of spreading disease as in the well-known case of tobacco mosaic. Special precautions are necessary to avoid this spread, such as disinfecta-

tion of tools and hands, and whenever possible the adoption of methods that reduce handling to a minimum.

Harvesting. The proper time of harvesting is of vital importance in reducing storage diseases, as is strikingly seen in potatoes. The danger of harvesting when the crop is wet with rain or dew is well illustrated in bacterial blight of beans and bacterial wilt of alfalfa, where this is the principal agency of spread of these diseases.

Storing. In all types of crops prompt and proper storage is necessary to avert destructive losses. In this the peculiarities of each crop and each disease must be considered especially as regards temperature and aeration. In general, cool temperatures and good ventilation, sometimes preceded by curing, are the requisites for storage disease control. Low humidities usually are most favorable for storage, but there are exceptions, as in protecting sweet potatoes from storage decays, in which high humidities are advisable. The combination of warm temperatures and inadequate ventilation is responsible for a variety of physiologic disorders, such as apple scald and potato black-heart, troubles that are combated by well-ventilated cool storage and in the case of fruits by oiling the fruits, packing them in oiled paper shreds, or wrapping them individually in oiled wrappers (Fig. 194).

Removal of Undesirable Plants or Plant Parts

Weed Control by Clean Culture. This aids in the control of those diseases that are favored by low vigor in the host plant so far as the competition afforded by weeds weakens the host. Also, the humid air and poor circulation in rank weedy undergrowth is a factor favoring those disease organisms that are dependent on ample moisture. In rare cases toleration of weeds may function in disease control, as in the case of heat canker of flax where the shading value of weeds may be of greater importance than their status as competitors.

Eradication of Wild Hosts. There are many cases in which weeds and other uncultivated plants are reservoirs of disease affecting nearby cultivated plants, examples of which are the harboring of potato and tomato viruses in wild *Solanaceae*, of grain diseases in wild grasses, and of fruit diseases in wild or neglected fruit trees. Spread of aster yellows from weeds into a crop field is shown in Fig. 217. Volunteer plants from a harvested crop are often the means of carrying a disease from one crop season to the next as is true of the cereal rusts and some of the spinach diseases. In these cases eradication of the wild plants often constitutes an important element of disease control.

Eradication of Alternate Hosts of Rust Diseases. In some cases, this procedure contributes to the control of the disease (stem rust of wheat in the northern Great Plains), and in other cases it may be fully

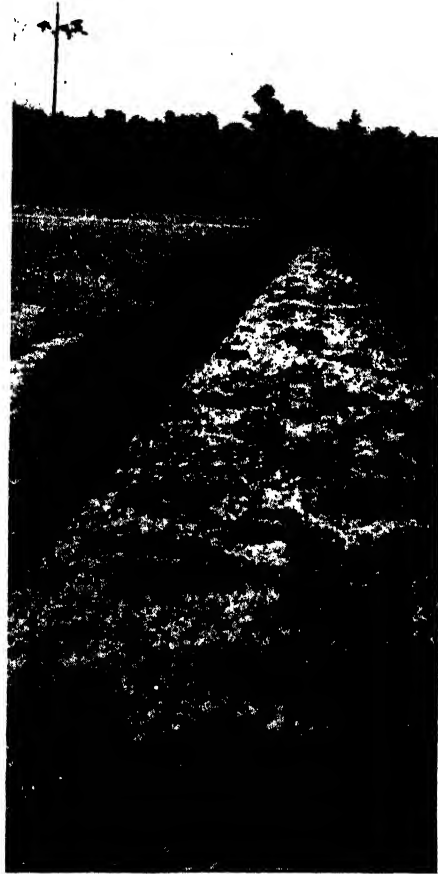


FIG. 217. Bed of endive, the yellowed plants affected with aster yellows, which has spread into the bed from affected weeds in the weedy strip at the left. This illustrates the role of weeds as sources of disease for cultivated crops. (Photograph, Cornell Univ. Agr. Exp. Sta.)

efficient in the complete control of the rust (stem rust in protected valleys, cedar-apple rust, pine blister rust). Eradication of the uredial host usually is more effective than eradication of the aecial host, since the aeciospores are unable to reinfest the host producing them, and in general are shorter-lived than urediospores.

Roguing. Roguing, or the destruction of individual diseased crop plants as soon as they appear, is the only effective control method for the virus diseases of stone fruits and is a valuable accessory control measure for the virus diseases of potatoes, cowpeas, and beans, bacterial wilt of cucurbits, and numerous other diseases. In these and other crops, roguing is worthy of more extensive use.

Removal of Infected Plant Parts. This procedure, with antiseptic treatment of the cut surfaces, is of greatest importance in connection with tree surgery and winter pruning of fruit trees. Even where the value of individual plants is much less, it is possible that prompt excision of infected parts might be used to advantage. In Asia Minor, where conditions of extremely low living standards prevail, highly effective disease control is associated with the practice of hand-picking individual diseased leaves of garden crops to salvage them for human or stock consumption. Here additional protection results from the burning of excreta and manure for fuel before using it as a fertilizer. While such extremes are not warranted under American conditions of living, they illustrate the efficiency of sanitation as a disease-control measure.

Removal of the Body or Reproductive Parts of the Pathogen. In a few instances, where the pathogen is large and conspicuous and the value of the host is considerable, a limited control value attaches to removal of the pathogen or its reproductive parts by hand. This applies in the removal of cedar-apple rust galls from ornamental cedars, of conks from fruit or ornamental trees, and the leafy mistletoe. Corn smut deserves more attention in this connection.

Protection of the Growing Crop with Fungicides

Sprays, dusts, and fumigants may be used to prevent the germination of spores that reach sprayed or dusted surfaces, and in the past this has been regarded as almost the only effect of such applications. But they have other functions. They may prevent an established fungus from producing spores, which probably is the explanation of the value of paradichlorobenzene fumigation of tobacco beds for control of downy mildew. Sprays may be used to kill the fungus on the ground before it reaches the crop, or on the plant, as in killing newly formed spores before they can become disseminated and produce infection. Ground spraying with an eradicant spray such as Elgetol or Sinox has become an important factor in controlling apple scab and other tree and bush fruit diseases, while part of the value of some protectant sprays for apple scab and other diseases appears to be a partial eradication of spores.

Bacteria being carried from plant to plant by water droplets, insects, or man often can slip past protective spray or dust coatings, down into the plant tissues, so we find that sprays and dusts are less helpful in controlling them than with fungus diseases. In rare instances, the mechanical protection or shading effect of sprays and dusts is used to advantage, as in the control of tipburn of potatoes. In certain crops, regular fungicidal spraying or dusting is essential to good culture (tree and bush fruits, potatoes and peanuts), in others it is not indispensable but helpful. As intensification of agriculture progresses, and cheaper and more efficient chemicals and better methods of application become available, a large field is opening in the further development of spraying and dusting.

SPRAYING VERSUS DUSTING: COMBINATION METHODS

Both spraying and dusting are used widely and each has advantages not pertaining to the other. It is often said that a good job of dusting is equal to a fair job of spraying. The effects of spraying are more lasting because sprays adhere to foliage longer than dusts. Dusting is easier and more rapid, takes less labor and motive power, is less likely to injure plants, and can be done by airplane or from the ground when it is so wet that the heavier spray equipment bogs down.

Recent developments in the application of fungicides and insecticides combine the advantages of spraying and dusting. One of these is the sprayer-duster which utilizes a high velocity air current, up to 250 m.p.h., as a carrier for concentrated dusts or liquids. Another type of equipment produces an artificial dew on leaves followed immediately by a dust application, which makes dusting practical at any time of day. A third innovation is the fog applicator which vaporizes small quantities of concentrated pesticide in hot air or steam; when this vapor comes in contact with cold air it condenses in a fine fog which covers the foliage. Still another new method is use of the air-blast sprayer in which a large volume of air, mixed with ordinary spray materials, blows the fluid as a fine mist into the foliage of trees, with a 50 to 80 per cent saving of labor compared with ordinary spraying. The aerosol method, releasing concentrated fungicides from Freon bombs or cartridges is finding use also, for example in protecting tobacco from downy mildew.

Where air is substituted for water or an inert dust carrier in diluting fungicides, by means of some of these newer types of equipment, the effectiveness of spraying can be obtained with the ease and economy of dusting, and in some cases an effective fungicidal coverage may be effected with a very small amount of fungicide per acre.

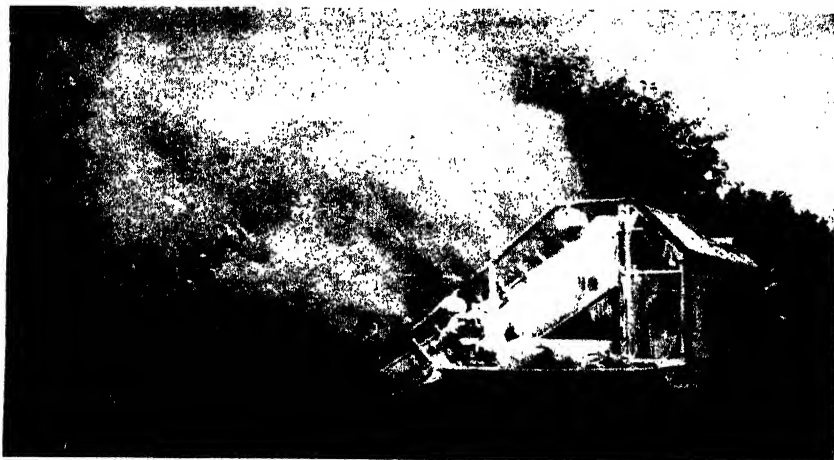


FIG. 218. (*Top*) A spray duster which applies wet dust to foliage at low velocity. This is an experimental model developed and photographed by the Dep. Entomology, New York State College of Agriculture at Cornell University.



FIG. 219. (*Bottom*) The speed-sprayer which discharges the spray liquid in a large volume of air at low pressure. Its advantages are reduction of mechanical injury to the crop, and rapid application with a small manpower requirement. With it 2 men can spray 30-40 acres of mature orchard a day, doing a better job with less effort than with hand-held spray guns. (Courtesy, John Bean Mfg. Co.)

SPRAYS AND SPRAYING

The principal fungicidal sprays and dusts, with common and trade names and uses, are listed in Table 9. Further particulars on some of these are given below.

Copper Series. Since its discovery and use by Millardet in 1882-1885, Bordeaux mixture has been the leading fungicide for grapes, tree fruits, potatoes, and many other crops. The various strengths used are expressed by a formula. For example, "Bordeaux 3-4-50" consists of 3 lbs. of CuSO_4 + 4 lbs. of unslaked lime (90 per cent CaO) or 6 lbs. of hydrated lime, in 50 gal. of water. An important function of the lime is to hold the copper in a form relatively unavailable and nontoxic to the host but toxic to parasitic fungi. Ready-mixed Bordeaux can be purchased or one can prepare quantities of stock solutions of 1 lb. per gal. of each chemical, and make the desired mixture by adding 1 gal. of the stock solutions, which keep well, for each pound specified in the formula. In mixing, strain the lime into the spray tank, add the water, and then pour in the copper sulfate, agitating well and using at once.

At present a number of comparatively insoluble or "fixed" copper compounds which are relatively noninjurious to foliage on this account, are receiving attention as substitutes for Bordeaux mixture. These, used as sprays or dusts, alone or more often with lime, are less likely to cause fungicidal injury than Bordeaux mixture, and therefore are particularly useful on plants which are very subject to spray injury, such as cucurbits and tomatoes.

Sulfur Series. Elemental sulfur (wetable sulfur) is a good fungicide and is less burning to foliage than some other spray materials. It is employed usually at the rate of 6 to 8 lbs. of finely divided sulfur in 50 gal. of water, made wettable or miscible by adding a proprietary wetting agent or a "spreader" such as calcium caseinate, oleic acid, glue, diatomaceous earth, flour, dextrin, skim milk, or dry lignin pitch, or by processes of manufacture which produce hygroscopic forms of sulfur. "Flotation sulfur" is very finely divided sulfur with certain impurities; it is a by-product of gas manufacture.

Lime-sulfur is one of the most useful of fungicides and is employed widely as an orchard spray except in hot weather when it is more likely to injure foliage than wettable sulfur or Bordeaux mixture. It is available commercially as a paste or a concentrated solution used at the rate of 2 to 5 gal. of solution per 100 gal. of water for spring spraying and at higher strengths for dormant spraying. The concentrated solution may be prepared on the farm by boiling quicklime (50 lbs.) and sulfur (100 lbs.) in water (enough to make a total of 50 gal.).

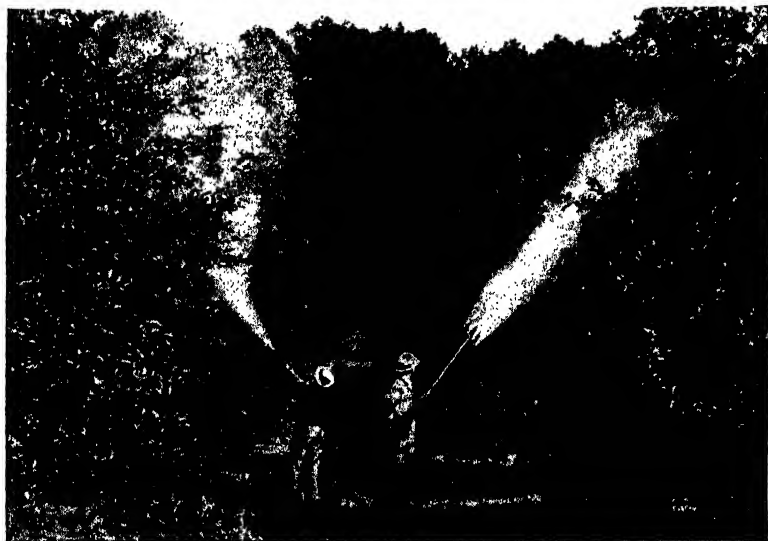
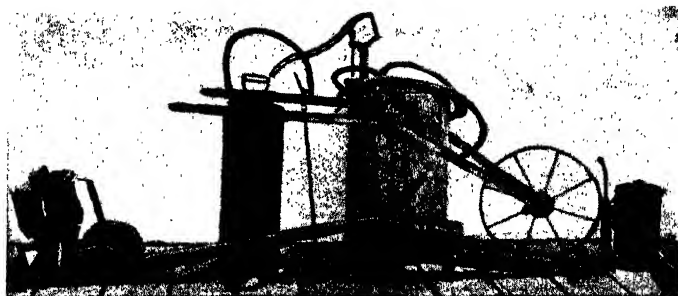


FIG. 220. (*Top*) Small hand-operated sprayers and dusters. (Photograph, Tenn. Agr. Exp. Sta.)

FIG. 221. (*Center*) Row sprayer applying Bordeaux mixture to potatoes at the rate of 200 gallons per acre. (Photograph, Tenn Agr. Exp. Sta.)

FIG. 222. (*Bottom*) A combination of rod and gun nozzles used in orchard spraying. (Photograph, Tenn. Agr. Exp. Sta.)

Organic Series. Recent years have witnessed remarkable advance in the development of numerous organic fungicidal spray materials, some of which are already supplanting copper and sulfur fungicides on important crops. These are often highly fungicidal but specifically so, being effective only against certain pathogens.

On apples, Fermate, Puratized, and Phygon are outstanding and have just about replaced sulfur materials. Fermate and Phygon are very promising for cherry, pear, and peach disease control. Dithane plus zinc-lime is noteworthy as a fungicide for early and late blight of potatoes, producing an important increase in yields, and Zerlate is highly effective against early blight. These fungicides also are very useful on tomatoes and celery. In tobacco seedbeds, Fermate and bismuth subsalicylate have become standard treatments for downy mildew control. Many other instances of the usefulness of the new organic fungicides are given in Table 9.

These fungicides are not expensive, considering their effective dilutions, are sometimes useful as dusts as well as sprays, show various degrees of compatibility with insecticides, and frequently have eradicant as well as protective fungicidal value. Many related compounds are being tested, and some of these promise to become important additions to the arsenal of weapons against crop disease.

Methods of Application. A long chapter could be devoted to the subject of sprayers and the technique of spraying but such a discussion is beyond the scope of this book. The subject is treated in detail by Mason, cited in the references at the end of this chapter.

Sprayers range from the hand atomizer through various types of hand and knapsack sprayers and barrel pumps up to large commercial power sprayers, and each has uses for which it is particularly suited (Figs. 220, 221, and 222). The art of spraying also requires knowledge of when and how to apply fungicides in order to accomplish the desired end with a minimum of cost and labor. Radically new types of sprayers and spray-dusters have been mentioned on p. 481.

DUSTS AND DUSTING

Sulfur alone or sulfur and lime mixtures are excellent dust fungicides. The sulfur should be so fine that 98 per cent of it will pass through a screen with 300 or 325 meshes to the inch and this fineness is obtained either by grinding or by precipitating sulfur from solutions. The finer dusts are less likely to burn foliage although there appears to be a limit of fineness beyond which nothing is gained. Sulfur alone rarely burns plants and may be combined freely with arsenical dusts without incompatibility. Sulfur dust has an important place in control of peanut leaf spot, diseases of tree

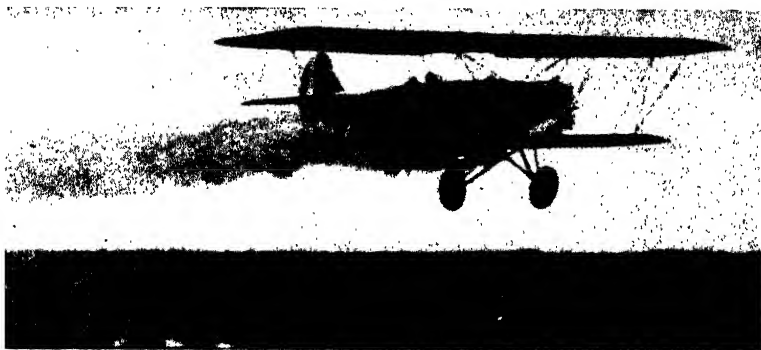


FIG. 223. Applying sulfur dust by airplane, for protecting wheat from rusts. (Courtesy, F. J. Greaney, Can. Dep. Agr.)

fruits, and powdery mildews of various crops. It is effective in controlling cereal rusts, but this use has not been developed on a commercial scale.

To maintain the fineness necessary in a good sulfur dust and prevent lumping, the dust frequently is treated with fillers or inert materials such as Fuller's earth, diatomaceous earth, talc, gypsum, hydrated lime, magnesium carbonate, or tricalcium phosphate. Such treated sulfurs are known as "conditioned sulfurs."

Copper-lime dust consists of copper compounds, usually of insoluble type, which are very finely ground and mixed with lime. The copper dusts are useful on tomatoes, melons, cucumbers, and other vegetables, and are sometimes used on potatoes.

Dusts may be applied with hand and knapsack type dusters, or on a large scale with power equipment. Recently, airplane dusting has developed at a rapid rate in North and South America, this being a cheap method of application when large acreages are involved (Fig. 223). Helicopters are particularly suitable for this work. To be most effective the dust should be applied before rains, and when the air is calm.

HAZARDS OF DUSTING AND SPRAYING

The usefulness of dusting and spraying is limited somewhat by dangers accompanying the practices. Chief of these are:

Spray Injury: 1. **BORDEAUX MIXTURE INJURY.** This injury appears as burns, shot-hole, yellowing, or defoliation of leaves, russetting of fruit, cankers and dieback of twigs, blighting of blossoms, and, in severe cases, general necrosis and death of the plant. Plants vary in their susceptibility to Bordeaux injury, the stone fruits being very susceptible, the pome fruits variable, and the potato fairly resistant. The greatest danger is at cool temperatures and when damp, foggy weather accompanies and

follows the spraying. It is believed that the reason for the injury is excessive carbon dioxide emitted under shady conditions, which dissolves in water on the leaves to form carbonic acid, which in turn liberates free copper from the copper sulfate, this entering the cells and killing them. The danger of Bordeaux injury is reduced by avoiding excessive applications, unusually strong solutions, or excessive copper sulfate in relation to the lime present, use of the more resistant varieties, and substitution of lime-sulfur for early spring applications or use of the recommended organic fungicides.

2. **LIME-SULFUR INJURY.** In contrast to Bordeaux injury, lime-sulfur injury occurs chiefly in hot weather. Leaves are burned, especially at the margins and tips, and the leaves of stone fruits may become shot-holed. In potatoes the plants are stunted and their life is shortened. The chemical appears to act on the chlorophyll in such a way as to starve the plants, leading to premature fruit drop. For prevention of lime-sulfur injury, do not use lime-sulfur on the most sensitive crops but substitute weak Bordeaux mixture or sulfur dusts or recommended organic fungicides, use the weakest effective concentration, spray moderately with a fine mist, and if an arsenical is added it should be arsenate of lead, not calcium or sodium, arsenate or Paris green.

3. **INCOMPATIBILITY OF SPRAYS.** While it is economical to combine different spray materials in the same application to control several pests at once, not all spray materials can be mixed without producing ineffective or injurious mixtures. For instance, oil and sulfur cannot be safely mixed. A chart of spray compatibility is given in Anderson and Roth, cited in the references at the end of this chapter. Of the newer fungicides, Fermate is compatible with spray oils but not with copper or lime-sulfur. Phygon and Dithane do not appear to be compatible with oils. On potatoes, Dithane-zinc-lime and Zerlate are compatible with DDT.

Poisoning of the Soil. In commercial orchards and vegetable acreages where intensive spray programs are followed year after year the problem of injurious accumulation of spray materials in the soil is a growing one. Already complaints of such soil poisoning have been heard in the apple-growing sections of Washington, Idaho, and New York, and in some cases it has become impossible to grow crops on such soils. The new organic fungicides are a boon to growers in this respect since they permit a shift away from the metallic fungicides used in past years, and the use of a greater variety of fungicides.

Spray Residues. On crops such as fruits and greens, spraying or dusting with poisons can constitute a menace to health. Growers or marketers can be prosecuted under the Pure Food and Drug Act if their



FIG. 224. Fumigating a tobacco seedbed with paradichlorobenzene. Crystals of the chemical are scattered over the regular seedbed cover, and then the bed is covered with a heavy cloth and wet down. (Courtesy, J. A. Pinckard, Va. Agr. Exp. Sta.)

products contain more than trace amounts of lead, arsenic, fluorine, or mercury. The use of the otherwise very effective Puratized for apple scab control is limited to early applications because of its mercury content. This danger can be avoided by the use of spray and dust materials which do not contain the poisonous metals, by avoiding excessive or late applications, and, in the case of fruit, by washing off spray residues in an acid bath, a practice regularly followed in apple production.

UNUSUAL CHEMICAL CONTROL PRACTICES

Fumigation. Although fumigants usually are directed against insects, there are cases where they are valuable aids in controlling plant diseases. The most notable case is the use of benzene vapor for the control of the very destructive downy mildew or "blue mold" in tobacco and cabbage seedbeds. The beds are covered with sheeting and the benzene allowed to evaporate from a free surface, unheated. Paradichlorobenzene also is used for this purpose, the method of use being illustrated in Fig. 224.

Sulfur vapor also is a good fungicide. In greenhouses it is sometimes the practice to coat steam pipes with sulfur paste. The slow volatilization serves as a preventive against such diseases as powdery mildew and black spot of roses. In much stronger concentration the vapor from boiling sulfur is used to fumigate sweet potato storage houses and other storage buildings. The vapor in this case is so highly injurious to plant tissues that the storage house must not be in use at the time of fumigation. Formaldehyde gas, which may be generated in high concentrations by adding potassium permanganate to formalin, is used for fumigation in a similar way. For soil fumigation see p. 463.

Fungicides in Irrigation Water. On a very limited scale use has been made of protecting plants by adding fungicides to irrigation water, as for downy mildew of spinach and tomato fruit rots.

Fungicidal Treatment of Bean and Hop Stakes and Twine. These stakes and twine have been found to harbor the overwintered pathogens of bean rust and downy mildew of hops, a source of danger which can be reduced or eliminated by dipping or spraying the stakes and twine with copper or sulfur fungicides, Puratized, or Phygon.

Absorption of Pesticides Through Roots. When selenium is added to soil it is taken up by plants and in some cases serves as an absorbed insecticide. Chrysanthemum plants have been made resistant to attack of the leaf nematode, at the same time sustaining little injury, by treating the soil with 25 p.p.m. of sodium selenate. Because of the toxicity of selenium to animals, the method cannot be used on food or feed crops. There is indication also that Dithane, mixed in soil, is taken up by plants, giving some protection against foliage diseases and insects.

Chemically Induced Bark Shelling. Scraping away the infected bark is a standard method of treating citrus trees infected with psorosis virus. By painting the lesions with DNOCHP (see Table 9) the trees are induced to shed their diseased bark, thus greatly reducing the labor of treating the lesions.

Cure of Diseased Plants

In human medicine great emphasis is laid on the application of curative remedies to individuals. In plants, on the other hand, the value of diseased individuals usually is small and not worth the cost of curing. Here the emphasis is on *preventing* disease (prophylaxis) although it has been seen that some plant protectants also act to eradicate disease organisms, in the fashion of therapeutic medicine. There are cases, however, in which the curing of individual plants may justify the cost either because of high value of the individuals or of low cost of the curative treatment, as in the following:

Heat Treatments of Plants for Nematode Control. Some types of nursery stock and greenhouse plants which are lightly infested with root knot can be cured by heating the root systems in water at 118°F. for 30 minutes. Not all plants will tolerate this temperature without injury, but it has been used successfully with black locust, Chinese elm, mulberry, cyclamen, begonia, peony and some roses. In the case of large lots of nursery trees the cost of the treatment has been about six cents per thousand. Many other plants affected with root knot should be tested for their heat resistance, as there is a probability of extensive use of this control

method on such stock. Heat treatments of this sort have been used with success in protecting coffee nursery stock from the root lesion (meadow) nematode.

Heat Treatments for Viruses. Kunkel has shown that peach trees suffering from peach yellows and some other virus diseases may be cured by heat treatments. It is questionable whether this would be practical with entire trees, but the work indicates the possibility of thus disinfecting peach budwood which might be of great value in producing virus-free stock and in the virus-free distribution of peach propagating material from one part of the country to another. Although some virus diseases cannot be cured in this manner (e.g., peach rosette and certain potato viruses), the field of usefulness of this method is largely unexplored.

Therapeutic Treatments of Trees. Individual shade trees may in some cases be worth several hundred dollars, and their value justifies attempts at curative treatments. Tree surgery is a step in this direction, although the effect of surgery is usually to postpone decay, not to cure it. Many attempts have been made to find chemicals that might be profitably injected into trees. Most of these attempts have failed but there appear to be a few authentic cases of cure of contagious disease in trees by injections.

In dealing with deficiencies of minor elements in trees, injection of salts is a reliable method of cure, and is widely practiced in some areas in combating chlorosis. But in view of the abuse of the practice by wandering charlatans posing as tree experts, the tree owner should have due caution in the use of such treatments.

With several systemic types of disease, such as *Verticillium* wilt of maple and eggplant, the Dutch elm disease, and the X-disease of peach, some success has been obtained in therapy by administering chemicals via the soil, by injection, or by dipping buds in the chemicals. The chemicals used include hydroxyquinoline compounds, pyrogallol, *p*-nitrophenol, hydroquinone, benzoic and ascorbic acids, malachite green, urea, and ammonia. Cure is frequently effected. A principal effect of these treatments is to inactivate, or provide an antidote to the toxins produced by the pathogens.

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Many of the state experiment stations and extension services issue disease control circulars and spray bulletins and calendars, and on application to your college of agriculture you will be furnished these.

Glossary

- Abiotic Disease.** A disease due to a nonliving cause.
- Acervulus, *pl.* Acervuli.** A nonsexual fungus spore mass, described on p. 188. and shown in Fig. 100.
- Acquired Immunity.** Immunity acquired during the lifetime of a plant.
- Acute Disease.** One that is rapid and destructive, often killing the plant in a short time.
- Aeciospores.** Rust spores formed in a cuplike aecium. Example: spores produced on barberry that carry infection to grain or grass plants.
- Aecium, *pl.* Aecia.** Cuplike fruiting body of rust fungi. (Figs. 7, 8.)
- Antheridium, *pl.* Antheridia.** Male sex organ in fungi. (Fig. 110.)
- Anthrachnose.** Any disease caused by fungi which produce nonsexual spores in acervuli.
- Antibiosis.** Antagonism between two organisms, particularly microorganisms in soil.
- Antibodies.** Chemical substances in the host that oppose the action of parasites, their products, or foreign materials.
- Apothecium, *pl.* Apothecia.** Cuplike ascus-containing fungus fruiting body. (Figs. 36, 60.)
- Appressorium.** A swelling on a fungus germ tube or hypha attaching it to a host tissue. (Fig. 4.)
- Ascocarp.** Any type of fungus fruiting body containing asci. (Fig. 36.)
- Ascospore.** Spore produced in an ascus. (Fig. 36.)
- Ascus, *pl.* Asci.** Saclike container of ascospores, usually 8. (Fig. 36.)
- Atrophy.** Underdevelopment or wasting away of a plant part.
- Autoclave.** Steam sterilizer.
- Autophyte.** A plant which manufactures its own food by photosynthesis and is not parasitic or saprophytic.
- Bacteriophage.** An ultramicroscopic virus which destroys bacteria.
- Bacteriostatic.** An agent which prevents the multiplication of bacteria without killing them, or term applied to this action.
- Basidiospore.** A spore produced on a basidium. (Fig. 29.)
- Basidium, *pl.* Basidia.** A spore-producing hypha, generally club-shaped and bearing four basidiospores. (Fig. 29.)
- Benign Disease.** One that does not cause severe injury to the host plant.
- Binucleate Cells.** Cells having two nuclei.
- Blasting.** Causing failure to produce fruit or seeds.
- Blight.** A disease causing general killing of leaves, flowers, stems, etc.
- Canker.** An open wound or dead lesion, often sunken, in a stem, surrounded by living tissues.
- Carrier.** An infected plant which shows no marked symptoms but is a source of infection for other plants.
- Causal Organism.** An organism (fungus, bacterium, nematode, etc.) which produces a given disease.
- Certification of Seed.** Seed production and marketing under control to maintain varietal purity and freedom from seed-borne pests.

Chemotherapy. Cure of diseased plants by chemical treatments.

Chlamydospore. A thick-walled, nonsexual resting spore formed by the rounding-off of any ordinary cell of fungus mycelium. (Fig. 68.)

Chlorosis. Yellowness of normally green tissues due to partial failure of chlorophyll to develop.

Chlorotic. Showing chlorosis.

Chronic Disease. One which persists in the host plant for a long period of time.

Cilium, pl. Cilia. A hairlike swimming organ on bacteria or zoöspores. (Fig. 110.)

Clamp Connections. Outgrowths of fungus hyphae which form bridges connecting hyphal cells. (Fig. 30.)

Cleistothecium, pl. Cleistothecia. A fruiting body containing asci and lacking any special opening, as in powdery mildews. (Fig. 36.)

Conidiophore. Hypha on which conidia are produced. (Fig. 37.)

Conidium, pl. Conidia. Any nonsexual spore except sporangiospores and chlamydospores. (Fig. 37.)

Contagious Disease. A parasitic disease that may be spread from one plant to another.

Continuous Spread of Disease. Gradual expansion of disease over an enlarging area by a series of short, plant-to-plant transmissions, involving plants uniformly through the area.

Control. Prevention of losses from plant diseases by any method.

Coremium, pl. Coremia. A cluster of erect hyphae bearing conidia. (Fig. 37.)

Court. See *Infection Court*.

Culturing. Artificial propagation of pathogenic or nonpathogenic organisms on nutrient media or living plants.

Damping-off. Seed decay in the soil or seedling blight, usually caused by soil fungi.

Diagnosis. Identification of the nature and cause of a disease.

Dieback. Progressive death of branches or shoots beginning at the tips. Characteristic of unhealthy trees.

Diploid. Having a double or 2N number of chromosomes.

Discontinuous Spread of Disease. Sudden carriage of disease to a new area without its affecting susceptible plants between the source of inoculum and the new point where disease is produced.

Disease. Any alteration of a plant that interferes with its normal structure, functions, or economic value.

Disinfectant. An agent that frees a diseased plant, organ, or tissue from infection.

Disinfected. Freed from infection by treatment with a disinfectant.

Disinfestant. An agent that kills or inactivates disease organisms before they cause infection of plants, as in the soil or on seed surfaces.

Disinfested. Successfully treated with a disinfestant.

Dissemination. The transport of inoculum from a diseased plant to a healthy one.

Endoconidium, pl. Endoconidia. Conidium formed within a hypha. (Fig. 83.)

Enphytotic Disease. One which is always present in a locality in relatively uniform amount.

Epiphyte. A plant which grows on another plant without being parasitic.

Epiphytology. The study of the relation of environment to the occurrence and character of disease.

Epiphytotic. The sudden and destructive development of a disease, usually involving an extensive area.

Eradicant Fungicide. One which destroys a fungus at its source.

Eradication. Control of a disease by eliminating the pathogen after it is already established. Eliminating potential host plants.

Etiolation. Yellowing of plants due to lack of light.

Etiology. Study of the causes of plant diseases and the nature of the causal agents.

Exanthema. Eruption or discharge of gum or other substances from diseased tissues.

Exclusion. Control of a disease by preventing its introduction into disease-free locations, as by quarantines.

Exudate. A liquid discharge from diseased tissues.

Facultative Parasite. An organism that is usually saprophytic but which, under some conditions, may become parasitic. Example: black bread mold.

Fasciation. A plant distortion, due to cell injury in the bud, resulting in flattened and sometimes spirally curved shoots.

Flag. A branch with dead leaves on an otherwise green tree.

Fructification. Production of spores by fungi. Fungus fruiting body or spore-bearing structure.

Fruiting Body. A complex fungus structure containing or bearing spores, as a mushroom, bracket fungus, perithecium, pycnidium, etc. (Figs. 28, 36, 37.)

Fumigant. A volatile disinfectant, the vapor of which destroys pathogens, insects, etc.

Fungicidal. Capable of killing or inhibiting fungi.

Fungicide. A chemical or physical agent which kills or inhibits fungi.

Fungistatic. An agent which prevents the development of fungi without destroying them, or term applied to this action.

Fungus, pl. Fungi. Organisms having no chlorophyll, with reproduction by sexual or asexual spores and not by fission, and usually with a mycelium having well-marked nuclei.

Gall. An outgrowth, often more or less spherical, of unorganized cells. (Fig. 150.)

Germ Tube. The hypha produced by a germinated fungus spore. (Fig. 4.)

Gram-negative, Gram-positive. Not being stained or being stained, respectively, by the Gram stain, used in classifying bacteria.

Hairy Root. Excessive, abnormal production of weak roots. (Fig. 150.)

Haploid. Having a single or N number of chromosomes.

Hauatorium, pl. Haustoria. (1) Special branch of a fungus hypha, especially one within a living cell, for absorbing food. (Fig. 4.) (2) A rootlike absorbing organ connecting a parasitic seed plant to the vascular system of its host.

Heteroecious Rust. One having different stages of its life cycle on two unlike types of host plants, as stem rust on cereals and barberry.

Heterothallic. Term applied to a fungus with sexes separate in different mycelia.

History of a Disease. The logically arranged historical facts relating to the disease itself, as distinguished from those relating more especially to the pathogen (Whetzel).

Homothallic. Term applied to a fungus with both sexes present in the same mycelium.

Host. Any plant attacked by a parasite.

Host Range. The various kinds of plants that may be affected by a given pathogen.

Hyaline. Colorless, transparent, or nearly so.

Hymenium. Spore-bearing layer of a fungus fruiting body, as the gills of a mushroom.

Hyperparasite. An organism that is parasitic on another parasite.

Hyperplasia. Production of an abnormally large number of cells.

Hyperplastic. Term applied to a disease in which an abnormally large number of cells are produced.

Hypersensitivity. Violent reaction of an organism to attack by a pathogen, with prompt death of invaded tissue preventing further spread of infection.

Hypertrophy. Abnormal increase in the size of cells.

Hypha, pl. Hyphae. A single thread of fungus mycelium.

Hypoplasia. Subnormal cell production.

Hypoplastic. Term applied to a disease in which there is subnormal cell production.

Immune. Exempt from disease; not subject to attack by a given pathogen.

Immunity. Freedom from disease due to the lack of qualities permitting, or to possession or acquirement of qualities preventing, the operation of the pathogenic factor.

Imperfect Fungus. One lacking any sexual reproductive stage.

Incubation. (1) The activities and developments of the pathogen during the incubation stage (Whetzel). (2) Maintaining inoculated plants or pathogens in an environment favorable for desired development.

Incubation Period. The period between inoculation of a plant and the first observed disease reaction.

Indexing. Determining the presence of disease in seed stocks, particularly potatoes, by preplanting portions of the stock.

Infect. To begin or produce disease.

Infection. (1) The process of beginning or producing disease. (2) A case of disease or presence of disease in a living plant (poor usage).

Infection Court. The place where an infection may take place (leaf, fruit, etc.).

Infection Stage. The period in a disease during which the host responds, symptoms appear, and the disease develops.

Infection Thread. A fungus hypha that initiates infection of a host; a germ tube.

Infectious. Term applied to a disease that may be communicated from one plant to another.

Infestation. Presence of disease in a population of plants, or of pathogens in a position or material where they have the possibility of producing disease, as in soil or on seed surfaces. Not to be confused with "infection" which can be applied only to living, diseased plants.

Inoculate. To place inoculum in an infection court.

Inoculation. The process of placing inoculum in an infection court.

Inoculum. Pathogens or parts of them which can infect plants, as spores, bacteria, fragments of mycelium.

Intercellular. Between cells.

Intracellular. Within cells.

Intumescence. A knoblike or pustulelike outgrowth, consisting of elongated cells, on leaves, stems, etc., caused by environmental disturbances.

Klendusity. Ability of a susceptible variety to escape infection under conditions in which other susceptible varieties contract the disease.

Lesion. A localized spot of diseased tissue.

Life History, Life Cycle. The complete succession of activities of a pathogen.

Local Infection. An infection involving only a limited part of a plant.

Medium, Culture. A solid or liquid nutrient substance on which organisms are grown, as agar or broth.

Micron. $\frac{1}{1000}$ millimeter; the usual unit for measuring spores, bacteria, and other microscopic objects.

Mildew. A plant disease in which the pathogen forms a fungus coating over the surface of plant parts; a fungus that causes such a disease.

Molds. Fungi with conspicuous mycelium or spore masses, usually saprophytes.

Monoecious Rust. A rust that can have all stages of its life cycle on a single species of plant.

Mosaic. Disease pattern of dark green and light green, yellow, or white on leaves of plants affected with certain virus diseases; virus disease that produces such a pattern.

Mummified. Dried up and shriveled, as in fruit affected by the brown rot pathogen.

Mummy. A dried, shriveled fruit, the result of certain fungus diseases. (Figs. 41, 44, 60.)

Mutation. A sudden variation in an organism which is transmitted to offspring.

Mutualistic. Term applied to a mutually beneficial relationship between organisms or symbiosis.

Mycelium, pl. Mycelia. A mass of fungus hyphae.

Mycology. The science dealing with fungi.

Mycorrhiza, pl. Mycorrhizae. Relationship of roots with symbiotic fungi, often necessary for normal growth of trees.

Natural Immunity. Immunity due to qualities inherent in a plant.

Necrosis, pl. Necroses. Death of plant tissues, as in rots, blights, and cankers.

Necrotic. Adjective form of necrosis; killing.

Nematocide. A chemical or physical agent that kills nematodes.

Nematodes; Nemas. Roundworms, or eelworms, causes of some plant diseases as root knot.

Obligate Parasite. A parasite that can develop only in living tissues, that has no saprophytic stage.

Oögonium, pl. Oögonia. Female sex organ in downy mildews and related fungi. (Fig. 110.)

Oöspore. A resting spore produced by sexual reproduction in the downy mildews and related fungi. (Fig. 110.)

Ostiole. A porelike opening of fruiting bodies such as pycnidia and perithecia.

Parasite. An organism that lives on or in a second organism, usually causing disease in the latter.

Pathogen. Any organism or factor causing disease.

Pathogenesis. The period in disease from the time of inoculation to the final reaction of the host.

Pathogenic. Capable of causing disease.

Pathogenicity. The ability of a pathogen to cause disease.

Pathology. The science of disease.

Pelleting. Coating seeds with a plastic substance in which a fungicide may be incorporated.

Perfect Stage. Sexual reproductive stage of a fungus.

Perithecium, pl. Perithecia. Flasklike ascus-containing fungus fruiting body. (Fig. 36.)

Pest. Term including any pathogen, insect, or other organism injuring plants or plant products.

Pesticide. Chemical or other agent that destroys pests, including fungicides, insecticides, nematocides, viricides, etc.

- Physiogenic Diseases.** Diseases caused by unfavorable environmental factors.
- Physiologic Races.** Pathogens of the same species and variety which are usually structurally indistinguishable but which differ in their physiologic behavior, particularly in their ability to parasitize varieties of a given host.
- Physiologic Specialization.** The occurrence of physiologic races within a species or variety of pathogen.
- Phytopathogenic.** Capable of causing plant disease.
- Phytopathology.** The science of plant disease.
- Predisposition.** Liability, susceptibility, or tendency to contract disease.
- Primary Infections.** The first infections by a pathogen after it has gone through a resting or dormant period.
- Proliferations.** Abnormal outgrowths.
- Promycelium, pl. Promycelia.** The basidium of smuts and rusts. (Fig. 17.)
- Provenience.** Origin; particularly the geographic origin of seed, inoculum, etc.
- Pseudomycorrhiza, pl. Pseudomycorrhizae.** Nonbeneficial or injurious association of a fungus with plant roots.
- Pustule.** A small, blisterlike elevation of the epidermis, often opening to expose a spore mass.
- Pycnidium, pl. Pycnidia.** Flasklike fruiting body containing conidia. (Fig. 37.)
- Pycniospore.** A spore from a pycnium in rusts, acting as a male gamete; spermatium.
- Pycnium, pl. Pycnia.** Sexual reproductive fruiting body in the rusts; spermatogonium. (Fig. 7.)
- Races.** See *Physiologic Races*.
- Range.** Geographic distribution. Not to be confused with "host range."
- Receptive Hypha.** Hypha in a pycnium of rusts with a female sexual function, fertilized by a pycniospore.
- Reinfect.** To produce infection again in a previously infected plant or organ.
- Resistance.** Ability of a host plant to suppress or retard the activity of a pathogen or other injurious factor.
- Resting Spore.** A spore, often thick-walled, that can remain alive in a dormant condition for some length of time, later germinating and capable of initiating infection.
- Rhizomorph.** A stringlike strand of fungus hyphae. (Figs. 33, 75.)
- Ring Spot.** A disease symptom characterized by yellowish or necrotic rings with green tissue inside the ring; virus diseases characterized by this symptom.
- Roguing.** Removal of undesired individual plants from a planting.
- Rosette.** A disease symptom in which stems are shortened producing a bunched growth habit; diseases in which this is a principal symptom.
- Russet.** Brownish roughened areas on the skins of fruit, from abnormal production of cork caused by diseases, insects, or spray injury.
- Rust.** (1) A fungus causing true rust, of the order Uredinales. (2) Disease caused by such a fungus. (3) Often loosely and improperly used for other types of diseases characterized by a rusty discoloration of plant parts.
- Saprogenesis.** That part of the life cycle of a pathogen in which it is not directly associated with a living host.
- Saprophyte.** An organism which feeds on lifeless organic matter.
- Saprophytic.** Referring to feeding on lifeless organic matter.
- Scab.** A crustlike disease lesion; a disease in which scab is a prominent symptom. (Fig. 40.)
- Sclerotium, pl. Sclerotia.** A resting mass of fungus tissue, often more or less spherical, usually not bearing spores. (Figs. 52, 54, 77.)

Scorch. A burning of tissues, the result of infectious or abiotic disease.

Secondary Infections. Infections resulting from inoculum from a primary infection or from other secondary infections, without an intervening inactive period.

Septate. Term applied to fungus hyphae or spores having cross-walls.

Septum, pl. Septa. A cross-wall in a fungus hypha or spore.

Serologic. Referring to blood tests used in identifying organisms or organic materials.

Seta, pl. Setae. A bristle occurring on or in some fungus fruiting structures. (Fig. 100.)

Shotholing. A disease symptom in which small roundish fragments drop out of leaves, giving them the appearance of having been riddled by shot. (Fig. 62.)

Sign. Any indication of disease other than a reaction of the host plant (symptom) such as structures of the pathogen, spores, mycelium, exudate, fruiting bodies, etc.

Slurry. A thick suspension of chemical used, for example, in coating seed with a fungicide.

Smuts. True smut fungi of the Ustilaginales or the diseases they produce.

Sorus, pl. Sori. A fungus fruiting structure or spore mass, especially spore masses of rusts and smuts.

Source of Inoculum. The place or object on or in which inoculum is produced.

Spermatium, pl. Spermatia. A spore from a pycnium (spermagonium) in rusts, acting as a male gamete; pycniospore.

Spermagonium, pl. Spermagonia. Sexual reproductive fruiting body in the rusts; pycnium. (Fig. 7.)

Sporadic. Term applied to a disease that breaks out occasionally without being constantly destructive.

Sporangiophore. Hypha bearing a sporangium. (Figs. 110, 111.)

Sporangiospore. A spore produced in a sporangium, particularly those produced by the black bread mold and related fungi. (Fig. 111.)

Sporangium, pl. Sporangia. An organ producing nonsexual spores within a more or less spherical wall. (Figs. 110, 111.)

Spore. A single- to many-celled reproductive body, in fungi and other lower plants, which may develop into a new plant.

Spore-mat. A spore-bearing layer of mycelium. (Fig. 76.)

Sporidium, pl. Sporidia. A basidiospore of a rust or smut fungus.

Sporodochium, pl. Sporodochia. A cluster of conidiophores interwoven together on a stroma or mass of hyphae.

Sporogenous. Spore-bearing.

Sporulate. To produce spores.

Spread of Disease. The propagation of disease, affecting increasing numbers of plants.

Staghead. Term applied to diseased trees in which there are leafless branches among and above normal leaf-bearing ones.

Sterigma, pl. Sterigmata. A projection from a hypha for supporting a spore. (Fig. 29.)

Sterilize. To remove or destroy all living organisms on or in an object or material.

Strain. An organism or group of organisms, differing in origin or in minor respects from other organisms of the same species or variety. Cf. *Physiologic Race*.

Stroma, pl. Stromata. A mass of fungus hyphae sometimes including host tissues, containing or bearing spores, and differing from a sclerotium in this respect. (Figs. 54, 55.)

Substrate. The substance or object on which an organism lives and from which it gets nourishment.

Summer Spore. A spore that germinates without resting, often short-lived, associated with rapid fungus increase during a favorable season.

Suscept. Any plant liable to infection by a given pathogen (Whetzel).

Susceptibility. The state or degree of being susceptible to disease.

Susceptible. Unable to oppose the operation of an injurious or pathogenic factor.

Symbiotic. The term applied to the relationship of two dissimilar organisms living together in close association; usually limited to cases in which the association is harmless or helpful to the two organisms.

Symptom. Any reaction of a host plant to disease.

Symptomatology. The science of symptoms of disease.

Syndrome. A group of signs and symptoms that occur together and characterize a disease.

Systemic. Term applied to a disease in which a single infection leads to general spread of the pathogen and its effects throughout the plant body.

Teliospores (Teleutospores). Spores of rusts, usually of a resting or winter type, which produce basidia on germinating, as the black spores of stem rust of cereals.

Therapeutic. Term applied to measures designed to cure diseased plants.

Therapy. Treatment to cure diseased plants.

Tolerant. Capable of sustaining disease without serious injury or crop loss.

Toxic. Poisonous.

Toxicity. Poisonous character of a substance.

Toxin. A poison formed by an organism.

Tylosis, pl. Tyloses. A cell outgrowth into the cavity of a xylem vessel, plugging it.

Uredial Stage. Stage in the life cycle of rusts in which urediospores or red rust spores are produced.

Urediospore (Uredospore, Urediniospore). Red rust or summer spores of rusts.

Uredium, pl. Uredia (Uredinium). Rust fruiting body producing urediospores (summer or red rust spores).

Vascular. Referring to the conducting system (xylem, phloem) of plants. Vascular infections often cause wilt diseases.

Vector. An agent (insect, man, etc.) which transmits disease.

Vein-banding. A symptom of virus diseases in which the regions along the veins of leaves are darker green than the tissue between the veins.

Viability. State of being alive.

Viricide. A chemical or physical agent that kills or inactivates viruses.

Virosis, pl. Viroses. A disease caused by a virus.

Virulence. Relative ability to cause disease.

Virulent. Highly pathogenic; with a strong capacity for causing disease.

Viruliferous. Virus-carrying; term applied particularly to virus-laden insects.

Virus. An obligately parasitic pathogen capable of reproduction in suitable hosts, ultramicroscopic, and recognizable principally or only by the visible effects produced in infected hosts.

Wilt. Loss of freshness and drooping of plants due to an inadequate water supply or excessive transpiration, or to vascular disease which interferes with utilization of water by a plant.

Witches' Broom. A disease symptom in which there is an abnormal brushlike development of many weak shoots.

Yellowing. Loss of green color due to degeneration or disorganization of chlorophyll or chloroplasts. Compare with *Chlorosis*.

Yellows. Term applied to diseases in which yellowing or chlorosis is a principal symptom.

Zoösporangium, pl. Zoösporangia. A sporangium which produces zoöspores.

Zoöspore. Spore capable of independent movement, as by swimming. (Fig. 110.)

Zygospore. Resting spore formed from the union of similar gametes, as in the sexual stage of the black bread mold. (Fig. 111.)

Index

The main discussion of a topic is indicated by *italic* numerals; illustrations, by **boldface** numerals.

A

- Acervuli, 94, **194**
- Acid, excessive, as cause of plant disease, 376
 - hydrochloric, as cause of injury to plants, 385
 - sulfurous, injury to plants, 383, 384
- Acidifying the soil, for damping-off, 243
- Acrogenus solani*, 331, **331**
- Aecia, 26, **30**
- Aeciospores, 26, **30**
- Agricultural chemistry, modern, work of von Liebig, 4
- Agriculture, intensification of, as origin of plant diseases, 429
 - philosophy of, modern, 11
 - plant disease in, significance of, 1-14
- Agrobacterium rhizogenes*, 292
- tumefaciens*, 291-294, **292**
- Agronomic practices, for leaf rust of wheat, 37
 - for stem rust of grains and grasses, 33
- Albugo candida*, 223, 224, **225**
- occidentalis*, 224
- Alfalfa, downy mildew, 222
 - leaf spot, 136
 - root rot, **157, 159**
 - rust, 40
 - white spot, 381
 - wilt, 285-287, **286**
 - control, 285
 - resistant varieties, 285
 - epiphytology, 285
 - etiology, 285
 - symptoms, 285
 - yellows, 373, **373**
- Alfalfa-clover, Southern, anthracnose, 195, **196**
- Algae, as cause of disease, 16
 - parasitic, 351
- Alkali, excessive, as cause of plant disease, 376, **377**
- Alternaria*, 246, **247**
 - solani*, 181-184, **182**
- Andropogon ternarius*, 120
- Anguina tritici*, 362, **363**
- Anthracnose, alfalfa-clover, Southern, 195, **196**
 - of beans, 194, **193-195**
 - control, 195
 - of cane fruits, 136, **137**
 - control, 137
 - of cereals, 188-192
 - control, 191
 - resistant varieties, 192
 - rotation, 191
 - sanitation, 191
 - seed treatment, 191
 - distribution and importance, 188
 - epiphytology, 191
 - etiology, 190
 - host plants, 189
 - symptoms and signs, 189, **190**
 - clover-alfalfa, Southern, 195, **196**
 - diseases caused by *Melanconiales*, 187-196
 - of flax, 195
 - of melons, 192, **192**
 - control, 193
 - of raspberry, black, 137
 - of tomato, 195
 - of wheat, **190**
- Anthocyanin, inhibition of pigment formation, as symptom of virus diseases, 302
- Antibiosis, 428
- Apbelenchoides*, 366
- Apothecium, 94
- Apple, bitter rot, 106, **107**
 - black rot, 105, **106**
 - blister canker, 108, **108**
 - blotch, 196-200, **198**
 - control, 200
 - exclusion, 200
 - fertilization, 200
 - pruning, 200
 - resistant varieties, 200
 - spraying, 200
 - epiphytology, 199
 - etiology, 199
 - history and distribution, 197
 - host plants, 197
 - importance, 197

Apple—(Continued)

blotch—(Continued)

symptoms and signs, 197

bark lesions, 199

fruit blotch, 199

leafspots, 197

browning, internal, 382

cork, external and internal, 373

dieback, 373

diseases of, 9

fire blight, 265-268, 267

powdery mildew, 129

rosette, 373

scab, 99-105, 100, 431

control, 102

applications, 103

spraying, 102-105

epiphytology, 102

etiology, 101

history and distribution, 99

host plants, 100

importance, 99

storage scab, 102

symptoms and signs, 100

scald, 382, 383

Arasan, 469, 474

for bunt of wheat, 54

for corn root, stalk, and ear rots, 257

for cotton seedling blight and boll rot complex, 250

for damping-off, 242

for smut, of onion, 72

of sorghum, 59

Arceuthobium spp., 346-350, 347, 348

campylopodium f. *divaricatum*, 348

Arthropods, as cause of plant disease, 15

Asci, 94

Ascomycetes, 17

fruiting bodies, 95

Ascospores, 94

Asparagus rust, 45, 45

Aspergillus, 254

niger, 245, 248

Asphyxiation, as cause of plant disease, 381

of fruits and vegetables in storage, 381

Asters, China, wilt, 154

yellows, 321-323, 322

control, 322

epiphytology, 321

etiology, 321

host plants, 321

symptoms, 321

B

Bacteria, as cause of plant disease, 16, 262-296

Bacterial blight, of barley, 280

Bacterial blight—(Continued)

of cotton, 275-279, 276

control, 277

economic importance, 276

epiphytology, 277

etiology, 277

history and distribution, 275

hosts, 276

symptoms and signs, 276

Bacterial canker, spot, and speck of tomatoes, 273, 274

control, 274

Bacterial diseases, of cereals, 279-282

control, 280

resistant varieties, 281

seed treatments, 280

epiphytology, 280

etiology, 280

of plants, control, 264

epiphytotic development, 264

pathology, 262

Bacterial hyperplasias, 291-296

Bacterial ring rot of potato, 287-289, 288

control, 288

etiology, 287

symptoms, 287

Bacterial spot of stone fruits, 268-270, 268

control, 270

etiology, 270

Bacterial wilt, 282-291

of corn, 262, 282-285, 282-284

control, 284

resistant varieties, 284

seed treatments, 284

epiphytology, 283

etiology, 283

symptoms, 282

of cucurbits, 290

control, 291

epiphytology, 291

etiology, 291

symptoms, 291

of muskmelon, 291

of sweet corn, 431

Bacteriology, of plant diseases, 262

Bacteriophage, 264

Bacterium solanacearum, 289

stewartii, 262, 282-285, 282-284

tomato, 275

Banana, Panama disease of, 7, 154

wilt, 154

Barbak, for damping-off, 242

Barbak-C, for corn root, stalk, and ear rots, 257

Barberry, common, eradication of, in control of stem rust, 33, 439

as host of stem rust, 24, 29, 30, 30

Japanese, immune from stem rust, 24, 30

- Bark shelling, chemically induced, 489
- Barley, bacterial blight, 280
Helminthosporium diseases, 177-189, 178
 leaf rust, 39
 net blotch, from *Helminthosporium teres*, 178, 179
 smut, 57
 covered, 66
 loose, 66
 spot blotch, from *Helminthosporium sativum*, 178, 179
 stripe, from *Helminthosporium gramineum*, 177, 178
- Barriers, for control of plant diseases, 458
 of Texas root rot, 163, 163
- Basidia, 74
- Basidiomycetes, 17
 as cause of plant diseases, 22-48
 characteristics of, 22
 clamp connections, 74, 75
 fleshy fungi, as cause of plant diseases, 74-86
 fruiting bodies, 75
 mycorrhizae, lack of, as cause of plant diseases, 86-93
 sexual reproduction, 75
 smuts, as cause of plant diseases, 49-72
- Basidiospores, 22, 49
- Basisporium gallarum*, 254
- Bean(s), anthracnose, 194, 193-195
 blight, 270-272, 271, 272
 mosaic, 316-318, 317
 control, 317
 etiology, 317
 symptoms, 316
 rust, 46, 46
 scalding of leaves, 392
- Beet(s), brown heart, 373, 374
 downy mildew, 222
 leaf spot disease, 186, 187
Verticillium wilt, 155
- Bermuda grass, downy mildew, 223
- Biology, work of Charles Darwin, 4
- Bitter rot, apple, 106, 107
 control, 108
- Black chaff of wheat, 281, 282
- Black gum, mistletoe, 347
- Black knot of plums and cherries, 110, 110
 control, 110
- Black raspberry, anthracnose lesions, 137
- Black root rot of tobacco, 166-169, 168, 417
 control, 169
 distribution and importance, 166
 epiphytology, 169
 etiology, 167
 suscept, 166
 symptoms, 166, 167
- Black rot, apple, 105, 106
 control, 106
- Black rust, of wheat, 27
- Black scurf of potatoes, 88-93, 90
 See also *Rhizoctonia* disease of potatoes
- Black spot of roses, 137, 138
 control, 138
- Blackheart of potatoes, 381, 382
- Blasting, of oats, 380, 381
 of sorghums, 381
- Blight, bacterial, of barley, 280
 of cotton, 275-279, 276. See also Bacterial blight of cotton
- bean, 270-272, 271, 272
 control, 271
- chestnut, 117, 426
- early, of potato, 181-184, 182
 control, spraying, 182
 of tomato, 181-184
 control, spraying, 182
- fire, of pear, 265-268, 266
- halo, of beans, 270-272, 271, 272
- late, of celery, 208, 208
- of potato, as cause of famine in Ireland, 1845-1847, 3
- of potato and tomato, 211-218, 214, 426, 431
 control, 216
 cultural methods, 217
 resistant varieties, 217
 spraying or dusting, 216
 storage, 217
 tuber selection, 216
- epiphytology, 216
 etiology, 215
 history and distribution, 21
 host plants, 213
 importance, 212
 symptoms and signs, 213
- Phacidium*, of conifers, 425
- Blister canker, apple, 108, 108
 control, 108
- Blister rust of five-needle pines, 43, 44
 control, 43
- Blossom infection smuts, 50, 61-67
- Blossom-end rot of tomato, 379, 380
- Blotch, apple, 196-200, 198. See also Apple blotch
 glume, of wheat, 205, 206
 net, of barley, from *Helminthosporium teres*, 178, 179
 spot, of barley, from *Helminthosporium sativum*, 178, 179
- Blue mold, of spinach, 8, 218, 219
 of tobacco, 220, 220, 221
- Boll rot, of cotton, 243-253
- Bordeaux mixture, 374, 483, 484
 for anthracnose, of beans, 195

- Bordeaux mixture—(Continued)
 for anthracnose—(Continued)
 of cane fruits, 137
 of melon, 194
 of sycamore, 112
 for apple blotch, 200
 for apple scab, 103
 for bitter rot, apple, 108
 for black knot of plums and cherries, 110
 for black rot, grape, 109
 for blight, early, of potato and tomato
 183
 late, of potato and tomato, 216
 for brown rot of stone fruits, 129
 for celery late blight, 208
 for cherry leaf spot, 135
 in control of plant diseases, 466
 for downy mildew, 222
 of cucurbits, 218
 of grapes, 222
 for fire blight of apple and pear, 268
 injury from, 486
 introduction of, 4
 for leaf spot, 201, 201
 elm, 111
 iris, 113
 for peach leaf curl, 141
 for powdery mildew of gooseberries, 129
 Boron, deficiency, as cause of plant disease, 373, 374
Botrytis, 187, 240
 Brand canker, of roses, 202
 Bread mold, 225–229
 Breeding for resistance to disease, 453
 bacterial, 264
 limitations, 454
 Brown canker, of roses, 202
 Brown heart, of beets, 373, 374
 of turnips, 373
 Brown rot of stone fruits, 129–134, 131
 control, 133
 dusting, 133
 measures supplementary to spraying,
 134
 spraying, 133
 epiphytology, 133
 etiology, 132
 history and distribution, 129
 host plants, 130
 importance, 129
 life cycle, 132, 132
 symptoms and signs, 130
 Brown spot, of corn, 229–232, 230, 231
 control, 231
 epiphytology, 231
 etiology, 230
 history and distribution, 229
 host plants, 229
 Brown spot, of corn—(Continued)
 importance, 229
 symptoms, 229
 of rice, 180
 Browning, internal, of apple, 382
 Bulb, nematode, 363
 Bunt of wheat, 50–57, 414
 control, 53
 dust treatment, 54, 55, 56, 57
 seed treatments, 53
 epiphytology, 53
 etiology, 53
 history and distribution, 50
 host plants, 51
 importance, 51
 symptoms and signs, 52, 52
 Bursting of fruits, 378, 379
 Bush fruits, *Verticillium* wilt, 155
- ### C
- Cabbage, clubroot, 234
 downy mildew, 222
Fusarium wilt, 443
 yellows, 151
 Cane fruits, anthracnose, 136, 137
 mosaic on raspberry leaves, 300
 orange rust, 45
 Canker(s), apple blister, 108, 108
 bacterial, of tomatoes, 273, 274
 blister, apple, 108, 108
 brand, of roses, 202
 brown, of roses, 202
 common, of roses, 201, 202
 cowpea, 272, 273
 crown, of roses, 202, 202
 graft, of roses, 201
 rose, 201–203, 202
 stem, 201
 Cantaloupe, powdery mildew, 453
 Carbon disulfide, for root knot, 360
 Care of the crop, 476–478
 choice of site, 476
 handling, 477
 harvesting, 478
 preparing the soil, 476
 seeding, 477
 soil amendment, 476
 storing, 478
 water supply, 477
 Carnation rust, 47, 47
 Carrot(s), downy mildew, 222
 root knot, 355
 Cedar apple rust, 41, 42
 control, 41
 Celery, blight, late, 208, 208
 control, 208
 cracked stem, 373

- Celery**—(Continued)
 leaf spot disease, 187
Cephaeleuros parasitica, 351
virescens, 351
Cephalosporium, 142, 143
diospyri, 155
 wilt, of elm, American, 154, 154
 control, 155
 slippery, 154
 control, 155
 of persimmon, 155
Ceratostomella ulmi, 115, 116, 117
Cercospora apii, 187
beticola, 186, 187
 diseases, 186
 leaf spot, of peanut, 185, 186
 of rice, 186
oryzae, 186
personata, 185, 186
Cereal(s), anthracnose, 188–192. *See also*
 Anthracnose, of cereals
 bacterial diseases, 279–282. *See also*,
 Bacterial diseases, cereals
 Gibberella seedling blight, 413, 413
 lodging of, 391, 391
 powdery mildew, 123–126, 124
 rust, 8, 26
 scab, 95–99, 413, 413
Ceresan, for cotton seedling blight and boll
 rot complex, 250
 for damping-off, 242
 New Improved, 471
 for bacterial canker, spot, and speck of
 tomatoes, 274
 for bunt of wheat, 54, 56
 for cotton seedling blight and boll rot
 complex, 252
 for smut, of oats and barley, 57
 of sorghum, 59
 for Victoria blight of oats, 181
Certification, in control of plant diseases,
 437
Charcoal rot, 259
Chemical substances, necessary, lack of, as
 cause of plant diseases, 368–375
Chemicals, for control of plant disease,
 470–475
 soil, excess of, as cause of plant disease,
 375–377
Chenopodium capitatum, 225
Cherry, black knot, 110, 110
 cracking, 378
 leaf spot, 134, 135
 control, 135
Chestnut blight, 117, 426
Chlamydospore(s), 17, 49, 49, 70, 143,
 168
Chlorine, as cause of injury to plants, 385
Chlorogenus callistephi, 321–323, 322
 var. *californicus*, 321
Chlorogenus persicae var. *vulgaris*, 335
 var. *micropersica*, 335
Chlorophyll, inhibition of pigment forma-
 tion, as symptom of virus dis-
 eases, 301
Chloropicrin, for black root rot of tobacco,
 169
 for damping-off, 242
 for root knot, 360
 for soil disinfestation, 464
 for wilt, watermelon, 150
Chlorosis, from excessive lime, 375, 376
Chytridiales, 211
Chytrids, 229–237
Cicadula sexnotata, 321
Citrus psorosis, 327–329, 328
 control, 327
 epiphytology, 327
 etiology, 327
 symptoms, 327
Cladosporium carpophilum, 184, 184
Cladosporium fulvum, 183, 184
Claviceps purpurea, 118–123, 119, 120
Cleistothecia, 94
Clover, dodder, 341
 downy mildew, 222
 rusts, 40
Clover-alfalfa, Southern, anthracnose, 195,
 196
Clubroot, of cabbage, 234
 of crucifers, 232–237, 417
 control, 235
 drainage, 237
 rotation, 235
 sanitation, 235
 soil disinfestation, 235
 varietal resistance, 237
 epiphytology, 235
 etiology, 234
 history and distribution, 232
 host plants, 233
 importance, 232
 symptoms and signs, 233
Cluster cups, 26, 30
Cobb scale, modified, for cereal rusts, 23
Cocoa, downy mildew, 222
COCs, for damping-off, 242
Coffee, downy mildew, 222
Cold injury, to plants, 387–390
 to harvested crops, 390
Coleosporium ipomoeae, 46
 solidaginis, 44
Collar rot, of tomato, 182
Collections, for study of plant diseases, 397
Colletotrichum, 195
 falcatum, 113

- Colletotrichum*—(Continued)
gossypii, 246
graminicolum, 188–192
lagenarium, 192, 192
lindemuthianum, 194, 193–195
lini, 195
phomoides, 195
trifolii, 195, 196
Common canker, of roses, 201, 202
Common scab, of potato, 294–296, 295
Conidia, 17, 94
 of *Helminthosporium*, 173
Conidiophores, 94
 of *Helminthosporium*, 173
Conifers, *Phacidium* blight, 425
Coniothyrium, 201–203
 fueckelii, 201
 wernsdorffiae, 201
Contagious plant disease(s), favored by
 soil, acid, 416
 alkaline, 417
 research, by Koch, 4
 by Pasteur, 4
Control of plant diseases, 433–441. *See*
 also individual diseases
 absorption of pesticides through roots,
 489
 artificial, 428
 chemically induced bark shelling, 489
 cultural methods, 457–490
 fumigation, 488, 488
 fungicidal treatment of bean and hop
 stakes and twine, 489
 fungicides, in irrigation water, 489
 laboratory tests, 407
 greenhouse and field tests of practices,
 407
 cultures, 408
 infestation, 408
 statistical significance of experiments,
 408
 investigation of, 399
 natural, 428
 antibiosis, 428
 hyperparasitism, 428
 procedures, 433
 regulation, 434–441
 removal of undesirable plants or plant
 parts, 478–480
 resistant varieties, 450–456
 accomplishments in development of,
 451
 breeding, limitations, 454
 genetics, 452
 need for, 450
 requirements, 450
 studies, 407–409
 unusual practices, 488
Copper, compounds, in control of plant
 diseases, 466
 deficiency, as cause of plant disease, 374
Copper-lime dust, 486
Copper-lime dusting, for downy mildew of
 cucurbits, 218
 for fire blight of apple and pear, 268
Corium solani, 331, 332
Cork, external and internal, of apples, 373
Corn, bacterial wilt, 282–285, 282–284.
 See also Bacterial wilt, corn
 brown spot, 229–232, 230, 231
 diseases of, 9
 Helminthosporium disease, 180
 potassium deficiency, 371
 rots in root, stalk, and ear, 253–257
 control, 256
 rust, 39
 smut, common, 67–71. *See also* Smut,
 common, of corn
 Stewart's disease, 282–285, 282–284
 wilt bacterium, 262
Corrosive sublimate, for *Rhizoctonia* control,
 92
Corticium vagum, 88–93, 90, 240
Corynebacterium insidiosum, 285–287, 286
 michiganense, 273, 274
 sepedonicum, 287–289, 288
Cotton, bacterial blight, 275–279, 276.
 See also Bacterial blight, cotton
 leaf curl, 8
 loss from diseases, 12
 root rot, 157, 158
 "rust," 371
 Texas root rot, 8, 156–166. *See also* Texas
 root rot, cotton
 Verticillium wilt, 155
 wilt, 416
Cotton seedling blight and boll rot com-
 plex, 243–253
 control, 249
 cultural measures, 252
 legal, 253
 resistant varieties, 252
 seed, clean, 253
 old, 252
 treatments, 249, 251
 epiphytology, 249
 etiology, 246
 importance, 243
 symptoms and signs, 244, 244, 245
Cotton wilt, 143–148, 145, 416
 control, 147
 epiphytology, 146
 etiology, 146
 history and distribution, 143
 host plants, 144
 importance, 144

- Cotton wilt—(Continued)
 symptoms and signs, 144
- Cowpea, canker, 272, 273
 scalding of leaves, 392
 virus disease, 301
 wilt, 154
- Cracked stem, of celery, 373
- Cranberries, keeping quality, 431
- Cronartium ribicola*, 43
- Crown canker, of roses, 202, 202
 control, 203
- Crown gall, 291–294, 292
 control, 293
 resistant varieties, 294
 epiphytology, 292
 etiology, 292
 symptoms, 292
- Crown rust of oats, 38
- Crucifers, clubroot, 232–237, 417. *See also*
 Clubroot of crucifers
 white rust, 223, 225
- Cryolite, for bacterial wilt of cucurbits,
 291
- Cryptosporrella umbrina*, 201
- Cucumber, *Verticillium* wilt, 155
- Cucurbits, bacterial wilt, 290
 downy mildew, 218, 218
 mosaic, 320, 320
 powdery mildew, 128, 128
- Culture media, in study of plant disease,
 400
- Cuprocide, for black spot of roses, 138
 for damping-off, 242
 for downy mildew of tobacco, 220
 for *Septoria* leaf spot of tomato, 208
- Cuprocide 54, for powdery mildew of
 roses, 127
- Cuprocide-sulfur-talc dust, for blight, late,
 of celery, 208
- Cuprocide-Y, for downy mildews, 222
- Cure of diseased plants, 489
 heat treatments, for nematode control,
 489
 for viruses, 490
 therapeutic treatment of trees, 490
- Curly top of sugar beet, 323–325, 323, 431
 control, 324
 epiphytology, 324
 etiology, 324
 host plants, 323
 symptoms, 323
- Cuscuta* spp., 340–346, 341–343
 approximata var. *urceolata*, 342
 campestris, 342
 epithymum, 342
 gronovii, 342
 indecora, 342
- Cyanide, in control of plant diseases, 466
- Cycle, grand, of disease, 427, 427
- Cylindrocladium scoparium*, 201
- ## D
- Dactylella bembicoides*, 367
- Damping-off, 238–243
 control, 241
 acidifying the soil, 243
 greenhouse and seedbed management,
 243
 postemergence treatments, 242
 seed treatments, 241
 soil disinfestation, 239, 242
 economic importance, 238
 epiphytology, 241
 etiology, 240
 host plants, 238
 symptoms, 238
 germination failure, 238
 postemergence, 239
 preemergence, 239
- Darlucal filum*, 37
- Darwin, Charles, work in biology and
 philosophy, 4
- DD mixture, for root knot, 360
- DDT, 487
 for apple, 103
 for smut of corn, 71
- De Bary, research on potato blight, 4
 work with stem rust, 23
- Dematiaceae*, 168, 172
- Development of resistant populations, 427
- Dibotryon morbosum*, 110, 110
- Dichlorisopropyl ether, for root knot,
 360
- Didymellina macrospora*, 113, 113
- Dieback, of apples, 373
- Dinitro, 475
 for bacterial spot of stone fruits, 270
 for citrus psorosis, 328
 for scab, apple, 104
- Diplocarpon rosae*, 137, 138
- Diplodia gossypina*, 244, 247, 247
 macrospora, 254
 zeae, 253, 255
- Disinfestation, by aging, 476
 by chemicals, 469
 in control of plant diseases, 437
 by heat, 475
- Distribution of plant diseases. *See individual*
diseases
- Dithane, 474, 485, 487
 for anthracnose of beans, 195
 for black rot, sweet potato, 115
 for blight, early, of potato, 182
 for brown rot of stone fruits, 134
 for downy mildew of tobacco, 220

- Dithane—(Continued)
 for leaf spot, cherry, 135
 for rust, carnation, 47
 for scab, apple, 103
 for *Septoria* leaf spot of tomato, 208
 Dithane-zinc-lime, 487
 Dithane-zinc sulfate-lime, for blight, late of
 celery, 208
 Dithane-ZnSO₄-lime spray, for blight,
 late, of potato and tomato, 217
Ditylenchus dipsaci, 363
 DN, for scab, apple, 104
 Dodder, 340-346, 341-343
 control, 344-346
 epiphytology, 344
 etiology, 342
 history and distribution, 340
 importance, 341
 symptoms and signs, 341
 in transmission of viruses, 307
Dorylaimus, 366
Dothiorella ulmi, 155
 Dow 6, for damping-off, 242
 Dow 9, 472
 for cotton seedling blight and boll rot
 complex, 250
 for damping-off, 242
 Dow 10, for damping-off, 242
 Dowfume, for root knot, 360
 Downy mildews, 211-223
 of alfalfa, 222
 of beet, 222
 of Bermuda grass, 223
 of cabbage, 222
 of carrot, 222
 of clover, 222
 of coffee and cocoa, 222
 of cucurbits, 218, 218
 control, 218
 of grains and grasses, 222
 control, 222
 of grape, 221, 222
 of hops, 222
 of lettuce, 222
 of lima bean, 222
 of onion, 222
 of pansy, 222
 of pea, 222
 of potato and tomato, 211-218, 214
 of rose, 222
 of soybean, 223
 of spinach, 218, 219
 control, 219
 of tobacco, 220, 220, 221
 control, 220
 of violet, 222
 Drainage, for control of clubroot of cruci-
 fers, 237
 Dry-land foot or root rot of wheat, 169-
 177
 control, 175
 rotation, 176
 epiphytology, 174
 etiology, 172, 173-175
 importance, 169
 symptoms and signs, 170, 171, 172
 Dry rot, of sugar beets, 373
 DuBay 1451, 1452, 1452F, 471
 DuBay 1452F, for cotton seedling blight
 and boll rot complex, 250
 for damping-off, 242
 Dusting, 485
 airplane, 34, 486
 for anthracnose, melon, 194
 for bacterial plant diseases, 264
 for black spot of roses, 138
 for blight, late, of potato and tomato,
 216
 for brown rot of stone fruits, 134
 for canker, crown, of roses, 203
 copper-lime, for downy mildew of cu-
 curbits, 218
 for downy mildew, of soybeans, 223
 of tobacco, 220
 hazards, 486-488
 seed, for bunt of wheat, 54
 for powdery mildew, of cucurbits,
 128
 for smut, of oats and barley, 57
 of sorghum, 59
 for *Septoria* leaf spot of tomato, 208
 vs. spraying, 481
 sulfur, for leaf rust of wheat, 37
 for stem rust of grains and grasses, 33,
 34
 for tomato leaf mold, 184
 Dusts, 485
 Dutch elm disease, 5, 6, 115, 116, 117, 426,
 490
 control, 116
- E**
- Edema, 379
 Eelworms as cause of plant diseases, 15,
 343-367
 Eggplant, *Verticillium* wilt, 490
 Elgetol, 475
 for anthracnose of cane fruits, 137
 for bacterial spot of stone fruits, 270
 for black rot, grape, 109
 for crown gall, 294
 for leaf curl, peach, 141
 Elm, American, *Cephalosporium* wilt, 154,
 154
 Dutch elm disease, 5, 6, 115, 116, 117,
 426, 490

Elm—(Continued)
 leaf spot, 110, 111
 control, 111
 phloem necrosis, 325–327. *See also*
 Phloem necrosis of elm
 slippery, *Cephalosporium* wilt, 154
Verticillium wilt, 155
Elsinoë veneta, 136, 137
 Embargoes, in control of plant diseases,
 437
Endoconidiophora fimbriata, 114, 115
 Endoconidiophore, 168
Endothia parasitica, 117
Entomophthorales, 211
Entyloma, 49, 72
 Environment, controlled, in study of plant
 diseases, 406, 406
 favorable to development of plant dis-
 ease, 412
 and parasitic disease, 412–417
 Epiphytes, 351
 Epiphytology, 403–407. *See also individual*
 plant diseases
 dissemination of diseases, 404
 of plant diseases, 424–432
 relation of disease to weather, 404
 seasonal development of disease, 403
 Epiphytotics, artificial, 407
 definition, 3
 from introduction of disease, 426
 from plant breeding, 427
 prediction of, 430
 from unnatural culture, 425
 from weather conditions, 425
 Eradication of hosts, alternate, of rust
 diseases, 479
 wild, 478, 479
 Ergot, fungus, reproductive structures,
 122
 of grains and grasses, 118–123, 119, 120
 control, 122
 epiphytology, 121
 ergotism, 122
 etiology, 120, 121, 122
 history and distribution, 118
 host plants, 119
 importance, 118
 symptoms and signs, 119
Erwinia amylovora, 265–268
 carotovora, 272
 tracheiphila, 290
Erysiphe graminis, 123–126, 124
 polygoni, 127
 Ethylene, as cause of injury to plants, 385
 bromide, for root knot, 360
 dichloride, for root knot, 360
 Etiology, of plant disease, 419–424. *See*
 also individual diseases

Eutettix tenellus, 324
 Evergreens, winter browning, 390
 Exclusion, for control of apple blotch, 200

F

Fallowing, in control of plant diseases, 458
 for downy mildews of grains and grasses,
 222
 Famine in Ireland, from Irish potato
 blight, 1845–1847, 3
 Fennel, 469, 474, 485, 487
 for anthracnose, beans, 195
 cane fruits, 137
 melon, 194
 tomato, 196
 for black rot, apple, 108
 grape, 109
 sweet potato, 115
 for black spot of roses, 138
 for black scurf of potatoes, 92
 for bitter rot, apple, 108
 for blight, early, of tomato, 182
 for blotch, apple, 200
 for damping-off, 242
 for downy mildew of tobacco, 220
 for leaf curl, peach, 141
 for leaf spot, cherry, 135
 for powdery mildew of roses, 127
 for rust, of carnation, 47
 of cedar and apple, 43
 for scab, apple, 103
 common, of potato, 296
 for *Septoria* leaf spot of tomato, 208
 for smut of onion, 72
 Fertility of soil in parasitic disease, 415
 Fertilization, for control of apple blotch,
 200
 Field crops, rusts of, 23–40
 Field observations of plant diseases, 393
 Fire blight of apple and pear, 5, 265–268
 control, 267
 epiphytology, 267
 etiology, 266
 history, distribution and importance, 265
 host plants, 265
 symptoms and signs, 265, 266
 Flag smut of wheat, 59
 Flax, anthracnose, 195
 rust, 40
 wilt, 8, 8, 150
 control, 151
 etiology, 151
Fomes applanatus, 76
igniarius, 76–81, 78, 80
 Formaldehyde, 469
 Fungal wilt, 287
 for cereal anthracnose, 191

- Formaldehyde—(Continued)
 for damping-off, 239, 242
 for fumigation of soil, 463, 463
 for *Rhizoctonia* control, 93
 for scab, common, of potato, 296
 for smuts, of barley, 57
 of oats, 57, 59
 for soft rot, sweet potato, and strawberry
 leak, 229
- Frost injury of fruits, 387, 389
- Fruit, frost injury, 387, 389
 spray injury, 486, 487
- Fruiting bodies, ascomycetes, 95
 of basidiomycetes, 75
 of fungi, 17
- Fumigants, organic, 465, 465
- Fumigation, for control of plant diseases,
 488, 488
 for downy mildew, of cabbage, 222
 of tobacco, 220
 of soil in control of plant diseases,
 463-466
 Bordeaux mixture, 466
 copper compounds, 466
 cyanide, 466
 formaldehyde, 463, 463
 mercury compounds, 466
 nonmetallic organic fungicides, 466
 organic fumigants, 465
 sulfuric acid, 466
 Tersan, 466
- Fungicides, in irrigation water, 489
 nonmetallic organic, 466
 for protection of growing crop, 480-489
 treatment of bean and hop stakes and
 twine, 489
- Fungus(i), 16-19
 as cause of plant disease, 16
 chestnut blight, 4
 classification, basis of, 17
imperfecti, 17
 infection by, 18, 18
 nutrition, 17
 sexual reproduction, 17
 spores, migration, study of, exploring
 upper air, 405
 structure, 16
- Fusarium*, 142, 143, 240, 243, 245-247, 254,
 256
 bataatidis, 151, 152
 conglutinans, 151
 culmorum, 97
 culmorum var. *cereale*, 97
 graminearum, 97
 hyperoxysporum, 151, 152
 infection of potatoes, 288
 lini, 150
 lycopersici, 148
- Fusarium*—(Continued)
moniliforme, 240, 246, 247, 254, 259
niveum, 7, 149
retusum, 148
vasinfectum, 143-148, 145
 wilt, 371
 in cabbage, 443
 of cucurbits, 290
 of potato, 153, 154
- Fusicladium dendriticum*, 102
- ### G
- Garden Dowfume, for root knot, 361
- Gas(es), illuminating, manufactured, as
 cause of injury to plants, 385
 poisonous, as cause of plant disease, 383
- Genetics of disease resistance in plants, 452
- Germination, spore, methods of studying,
 398
 tests, for corn root, stalk, and ear rots,
 256
- Gibberella* seedling blight of cereals, 413,
 413
- Gibberella zeae*, 95-99, 254, 256
- Gloeosporium*, 188
- Glomerella*, 190
 cingulata, 106, 107
 gossypii, 244, 246
- Glume blotch of wheat, 205, 206
- Gnomonia ulmea*, 110, 111
 veneta, 112, 112
- Golden nematode, of potato, 7, 364
- Gooseberries, mildew, powdery, 129
- Gossypium thurberi*, 276
- Graft canker, 201
- Grains, stem rust, 23-40
- Grapefruit, chlorosis, 375
- Grapes, black rot, 108, 109
 control by spraying, 109
 downy mildew, 221, 222
- Grasses, rusts, 40, 41
 stem rust, 23-40
- Gray molds, 186
 control, 187
 of onions, 187
- Guignardia bidwellii*, 108, 109
- Gymnoconia interstitialis*, 45
- Gymnosporangium clavipes*, 41
 germinale, 41
 globosum, 41
 juni-peri-virginianae, 41, 42
- ### H
- Halo blight, of beans, 270-272, 271, 272
 of oats, 279
- Handling, in control of plant diseases, 477

- Harvesting, in control of plant diseases, 478
- Haustorium(a), 19
- Heat, in disinfestation of soil, 459-463, 460-462
- cankers of seedlings, 386
- injury to plants, 386
- treatment, of plants for nematode control, 489
- for viruses, 490
- Helminthosporium carbonum*, 180
- Helminthosporium*, in corn, 180
- diseases of barley, 177-180, 178
- gramineum*, 177, 178
- oryzae*, 180
- sativum*, 169-177, 178, 179
- terres*, 178, 179
- turcicum*, 180
- victoriae*, 180
- Helotiales*, 94
- Heterodera marioni*, 353-361, 355, 356
- rostochiensis*, 364
- schachtii*, 361, 362
- Higginsia hiemalis*, 134, 135
- History of plant diseases. *See individual diseases*
- Hollow heart, of potatoes, 379
- Hollyhock rust, 47, 48
- Hopperburn, of potatoes, 387, 388
- Hops, downy mildew, 222
- Verticillium* wilt, 155
- Hordeum pusillum*, 41
- Host plants, of plant diseases, 18. *See also individual diseases*
- reduction in population, 427
- Host ranges, of viruses, 307
- Hymenium, 74
- Hymenomycetes, 74
- Hyperparasitism, 428
- Hypersensitivity in control of plant diseases, 448
- Hypha(ae), 16
- Hyphomycetes, as cause of plant diseases, 142-187
- I**
- Immunity, acquired, of viruses, 309, 309, 310
- to plant diseases, 445-450
- acquired, 448
- Immunology, plant, basic concepts, 443-445
- Imperfect fungi, as cause of plant diseases, 142-208
- Importance of plant diseases, 1-13. *See individual diseases*
- Infection by fungi, 18, 18
- Infectiousness of viruses, 303
- Inoculation, of a pathogenic organism, 420
- continuous spread, 421, 422
- discontinuous spread, 422
- incubation period, 422
- infection stage, 423
- of plants, for study of disease, 402
- Insects, as carriers of viruses, 306
- Inspection in control of plant diseases, 437, 438
- Intumescence, 379
- Ipomoeahederacea*, 46
- Iris leaf spot, 113, 113
- control, 114
- Irish potato blight, as cause of famine in Ireland, 1845-1847, 3
- devastation in Europe, United States, and Canada, in 1845, 3
- Iscobrome, for root knot, 360
- Isothan, for black rot, sweet potato, 115
- for leaf spot, cherry, 135
- Isothan Q15, 472
- for scab, apple, 103
- J**
- Jimson weed, virus disease, 301
- K**
- Kelsey spot of plums, 386, 387
- Koch, Robert, and research on contagious disease, 4
- Koch's postulates, 399
- L**
- Larvacide, for damping-off, 242
- for root knot, 360
- Latent mosaic virus, potato, 329
- Leaf curl, of cotton, 8
- of peach, 138-141, 139
- Leaf drop, cranberry, 477
- Leaf mold, of tomato, 183, 184
- Leaf roll virus, of potato, 331, 332
- Leaf rust, of barley, 39
- of wheat, 34-37, 36, 431
- control, 37
- agronomic practices, 37
- biologic, 37
- resistant varieties, 37
- sulfur dusting, 37
- epiphytology, 36
- etiology, 35
- host plants, 35
- life cycle, 36
- symptoms and signs, 35
- Leaf spot, alfalfa, 136
- angular, of tobacco, 275

- Leaf spot—(Continued)
 of beets, 186, 187
 of celery, 187
Cercospora, of peanut, 185, 186
 of rice, 186
 cherry, 134, 135
 elm, 110, 111
 iris, 113, 113
Septoria, of tomato, 206–208, 207
 sorghum, 279, 280
 Leaf streak, sorghum, 279, 280
 Leaf stripe, sorghum, 279
 Leak, strawberry, 225–229
 Legumes, powdery mildew, 127
 Lettuce, downy mildew, 222
 Lichens, as cause of disease, 16
 Liebig, von, and modern agricultural chemistry, 4
 Light, excessive, as cause of plant diseases, 391
 insufficient, as cause of plant diseases, 390
 in parasitic disease, 415
 unfavorable, as cause of plant diseases, 390–392
 Lima bean, downy mildew, 222
 Lime, chlorinated, for *Rhizoctonia* control, 93
 excessive, as cause of plant disease, 375, 376
 Lime-sulfur, injury from, 487
 for spraying, 483
 Little peach, 335
 Local infection smuts, 50, 67–72
 Lodging of cereals, 391, 391
 Losses, from plant diseases, 9
Lycopersicon pimpinellifolium, 148

M

- Macrophomina phaseoli*, 240, 246, 248, 254, 258, 259
Macropsis trimaculata, 306, 335
 Magnesium, deficiency, as cause of plant disease, 374
 Manganese, deficiency, as cause of plant disease, 374, 375
 Manuring, for control of Texas root rot, 164, 165
 Maple, diseases of, 9
 Verticillium wilt, 155, 490
Marmor cucumeris, 320, 320
 var. *upsilon*, 329
 dubium, 329
 lacerans, 337
 persicae, 336, 337
 phaseoli, 316–318, 317
 sacchari, 319

- Marmor*—(Continued)
 solani, 330
 tabaci, 313–316, 314
Marssonina populi, 196
Melampsora lini, 40
Melanconiales, as cause of anthracnose diseases, 187–196
 Melons, anthracnose, 192, 192
 Mercuric chloride, for bacterial canker, spot, and speck of tomatoes, 274
 Mercurochrome, for black scurf of potatoes, 88
 Mercury, acid, for *Rhizoctonia* control, 92
 bichloride, for bacterial wilt of corn, 284
 compounds, in control of plant diseases, 466
 dusts, organic, for bacterial wilt of corn, 284
 soil treatment, for clubroot of crucifers, 236
 yellow oxide of, for *Rhizoctonia* control, 92
 Methasan, for bitter rot, apple, 108
 for blight, early, of potato, 182
 for leaf spot, cherry, 135
 Methyl bromide, for root knot, 360
 Migration of fungus spores, study of, exploring upper air, 405
 Mild mosaic virus, of potato, 330
 Mildew, downy. *See* Downy mildew
 powdery. *See* Powdery mildew
 Millardet, and introduction of Bordeaux mixture, 4
 Milo disease, 257, 258
 Mistletoe, 346–350, 347, 348
 control, 350
 in forest trees, 350
 in ornamental and shade trees, 350
 epiphytology, 349
 etiology, 349
 history and distribution, 346
 host plants, 347
 importance, 347
 symptoms and signs, 347
 Moisture, in parasitic disease, 414
 Mold, blue, of spinach, 8, 218, 219
 of tobacco, 220, 220, 221
 bread, 225–229
 Molluscs, as cause of plant disease, 15
Moniliales, as cause of plant diseases, 142–187
Monilinia fructicola, 129–134, 131
Mononchus papillatus, 366
 Mosaic, bean, 316–318, 317. *See also* Bean mosaic
 cane fruit, on raspberry, 300
 cucurbit, 320, 320
 mild, of potato, 330

Mosaic—(Continued)

- peach, 336, 337
- rugose, of potato, 329, 330
- sugar cane, 319
- tobacco, 313–316, 314. *See also* Tobacco mosaic
- wheat, 318, 318
- yellow, 318
- Mucilago spongiosa*, 236, 237
- Mung bean, root knot, 355
- Muskmelon, bacterial wilt, 291
- Verticillium* wilt, 155
- Mustard, white rust, 224
- Mutability, of viruses, 308
- Mycelium, 16
- Mycorrhizae, 86–93
 - deficiency, 87
 - ectendotrophic, 86
 - ectotrophic, 86, 86
 - endotrophic, 86
 - function, 87
 - fungi, 87
 - pseudo-, 86
- Myzus persicae*, 330, 337

N

- Nanus mirabilis*, 335
- National Plant Quarantine Act of 1912, 5, 434
- Neck rot of onions, 187
- Necroses, bacterial, 265–282
- Nemacides, soil, 464
- Nematode(s), biologic control, 366
 - bulb, 363
 - as cause of plant disease, 15, 343–367
 - control, heat treatment of plants, 489
 - golden, of potato, 364
 - physiologic specialization, 366
 - root lesion, 364, 365
 - rye, control, 362
 - stem, 363
 - sugar beet, 361, 362
 - wheat, control, 362
 - and rye, 362, 363
- Net blotch, barley, from *Helminthosporium teres*, 178, 179
- Nicotine, for smut of corn, 71
- Nicotinia glutinosa*, 305, 305, 315, 316
- Nitrogen, deficiency, as cause of plant diseases, 368
 - excessive, as cause of plant disease, 375
- Nummularia discreta*, 108, 108

O

- Oats, blasting, 380, 381
- crown rust, 38

Oats—(Continued)

- halo blight, 279
- Helminthosporium* disease, 180
- smuts, 57, 58
- Obligate parasitism, of viruses, 303
- Oil heaters, for protection of fruit trees from frost, 389
- Okra, *Verticillium* wilt, 155
- Onion, downy mildew, 222
 - gray mold, 187
 - smudge, 195
 - smut, 72, 72
- Oöspore, 210, 211.
- Orange rust of cane fruits, 45
- Organisms, identification of, in study of plant diseases, 398
 - isolation of, in pure culture, 400
 - pathogenic, history of, 420
 - life-history of, 420
 - names of, 419
- Origin of plant diseases, 429
 - authentic new diseases, 430
 - improved recognition, 430
 - increasing proportion of susceptible hosts in population, 429
 - intensification of agriculture, 429
 - wider distribution of old diseases, 429
- Ornamentals, rusts of, 47–48
- Over-production in agriculture, control of, 12
- Oxygen, lack of, as cause of plant disease, 381

P

- Panama disease of banana, 7, 154
- Pansy, downy mildew, 222
- Parasites, obligate, 19
 - seed plant, 341–351
- Parasitic disease, effect of temperature, 413, 413
 - and environment, 412–417
 - moisture, 414
 - soil, 415
 - wind, 415
- Parasitic flowering plants, as cause of plant disease, 15
- Parasitism, types of, 19
- Pasteur, Louis, and research on contagious disease, 4
- Pathogenesis, 420
- Pathogens, 18
- Pathology, of bacterial plant diseases, 262
- Pea, downy mildew, 222
 - wilt, 154
- Peach, brown rot, 131
 - leaf curl, 138–141, 139
 - control, 141

Peach—(Continued)

leaf curl—(Continued)

- epiphytology, 140, 140
- etiology, 140
- history and distribution, 138
- host plants, 139
- importance, 138
- symptoms and signs, 139

little, 335

mosaic, 336, 337

phony, 335

root knot, 155

scab, 184, 184

control, spraying, 185

Texas root rot, control, barrier, 163

victim of new virus disease, 7

X-disease, 337, 490

yellows, 302, 335. *See also* Yellows, peach

Peanut, *Cercospora* leaf spot, 185, 186

Pear, fire blight, 265-268, 266

Pecan, rosette, 372, 372

Pellicularia filamentosa, 88-93, 90

Penicillium, 18, 254

Pentachlorethane, for root knot, 360

Peppers, *Verticillium* wilt, 155

Perisporiales, 94

Perithecium, 94

Peronospora effusa, 218, 219

tabacina, 220, 220, 221

Persimmon, *Cephalosporium* wilt, 155

victim of fungus, 5

Pestalotia, 196

Pesticides, absorption of, through roots, 489

Pezizales, 94

pH requirements, 416, 416

Phacidium blight of conifers, 425

Phloem necrosis of elm, 325-327

control, 326

epiphytology, 326

etiology, 326

hosts, 326

importance, 326

range, 326

symptoms, 326

Phoradendron spp., 346-350, 347, 348

Phosphorus, deficiency, as cause of plant diseases, 370, 370

Phycomycetes, 17

as cause of plant diseases, 210-237

Phygon, 469, 473, 485, 487

for anthracnose of beans, 195

for bitter rot, apple, 108

for black rot, sweet potato, 115

for black scurf of potatoes, 92

for black spot of roses, 138

for blight, late, of celery, 208

of potato and tomato, 217

Phygon—(Continued)

for brown rot of stone fruits, 134

for corn root, stalk, and ear rots, 257

for damping-off, 242

for downy mildews, 222

for leaf spot, cherry, 135

for scab, apple, 103

for *Septoria* leaf spot of tomato, 208

Phyllosticta, leaf spots of other hosts, 201, 201

solitaria, 196-200, 198

Phymatotrichum omnivorum, 156-166

Physalospora obtusa, 105, 106

tucumanensis, 112

Physiogenic plant diseases, 15, 368-392

Phytophthora, 240

infestans, 211-218, 214

Pine, five-needle, blister rust, 43, 44

Plant disease(s), due to unfavorable air conditions, 381-385

due to ascomycetes, 94-141

due to bacteria, 262-296

due to basidiomycetes, 22-48

fleshy fungi, 74-86

mycorrhizae, 86-93

smuts, 49-72

causes, investigation of, 399-403

control, 433-441

artificial, 428

by inducing resistance, 442-456

natural, 428

studies, 407-409

environment favorable to development of, 412

epiphytology, 424-432

etiology, 419-424

fight against, 13

due to hyphomycetes (*Moniliales*), 142-187

due to imperfect fungi, 142-208

due to lack of necessary chemical substances, 368-375

due to light, excessive, 391

insufficient, 390

unfavorable, 390-392

losses from, 9

methods of study, 393-410

collections, 397

field observation, 393

literature review, 409

organisms, identification of, 398

specimens, laboratory examination, 397

preservation of, 397

surveys, 394-397, 396

due to nematodes or eelworms, 343-367

a new science, 2

due to nitrogen shortage, 368

origin, 429

Plant disease(s)—(Continued)

- due to parasitic or predatory animals, 15
- due to parasitic seed plants and algae, 340-351
- due to pathogenic plants, 15
- due to phycomycetes and related fungi, 210-237
- physiogenic, 368-392
- relation to weather, 404
- due to rusts, 22-48
- significance of, in agriculture, 1-14
- due to soil conditions, unfavorable, 368-381
- due to *Sphaeropsidales*, 196-208
- study of, in controlled environment, 406, 406
- due to temperatures, unfavorable, 386-390
- types of, 15-16
- due to viruses, 15, 298-338
- due to water deficiency, 377-379
- Plant pathology, modern, birth of, 4
- as new science, 13
- Plasmodiophora brassicae*, 232-237
- Plasmopara viticola*, 221, 222
- Plums, black knot, 110, 110
- Kelsey spot, 386, 387
- Podosphaera leucotricha*, 129
- Poisoning of soil by sprays, as cause of plant disease, 377
- Potassium, deficiency, as cause of plant diseases, 371, 371
- Potato(es), bacterial ring rot, 287-289, 288
- blackheart, 381, 382
- black scurf, 88-93, 90
- blight, early, 181-184, 182
- late, 3, 211-218, 214, 426, 431
- research by de Bary, 4
- diseases of, 9
- Fusarium* infection, 288
- Fusarium* wilt, 153, 154
- hollow heart, 379
- hopperburn, 387, 388
- introduction to Europe, 3
- leaf roll virus, 331, 332
- mosaic, latent, 329
- mild, 330
- rugose, 329, 330
- nematode, golden, 7, 364
- purpletop wilt, 7
- Rhizoctonia* disease, 88-93, 90
- scab, common, 294-296, 295, 417
- spindle tuber virus, 331, 331
- tipburn, 387, 388
- vein-banding virus, 329
- Verticillium* wilt, 155
- virus diseases, 329-334
- yellow dwarf, 7, 333
- Powdery mildew, of apple, 129
- control, 129
- in cantaloupe, 453
- of cereals, 123-126, 124
- control, 126
- resistant varieties, 124
- sulfur dusting, 126
- epiphytology, 125
- etiology, 125
- history and distribution, 123
- host plants, 123
- importance, 123
- symptoms and signs, 124
- of cucurbits, 128, 128
- control, 128
- as destroyer of wine industry in Madeira, 3
- of gooseberries, 129
- control, 129
- of legumes, 127
- control, 128
- of roses, 126, 127
- control, 127
- spraying, 127
- sulfur dust, 127
- Pox, of sweet potato, 417
- Pratylenchus pratensis*, 364, 365
- Promycelium, 49
- Protozoa, as cause of plant disease, 15
- Pruning, for control of apple blotch, 200
- Pseudomonas andropogoni*, 279
- angulata*, 275
- coronafaciens*, 279
- phaseolicola*, 270-272, 271, 272
- solanacearum*, 289
- syringae*, 279
- tabaci*, 275
- Pseudoperonospora cubensis*, 218, 218
- Pseudopeziza*, 190
- medicaginis*, 136
- Puccinia anomala*, 39
- antirrhini*, 47, 47
- asparagi*, 45, 45
- coronata*, 38
- dispersa*, 39
- glumarum*, 38. See also Stripe rust of wheat and other gramineae
- graminis*, 23-40. See also Stem rust
- avenae*, 24
- secalis*, 24
- tritici*, 24
- helianthi*, 40
- malvacearum*, 47, 48
- peridermiospora*, 41
- purpurea*, 39
- sorgbi*, 39
- trititica*, 34-37. See also Leaf rust of wheat

- Puritized, 472, 485
 - for anthracnose of beans, 195
 - for bitter rot, apple, 108
 - for black rot, sweet potato, 115
 - for black scurf of potatoes, 92
 - for downy mildews, 222
 - for scab, apple, 103
- Purpletop wilt, of potato, 7
- Pycnidia, 94
- Pycnium(a), 26
- Pythium*, 243, 255
 - arrhenomanes*, 254, 259
 - debaryanum*, 238-243
 - ultimum*, 226

Q

- Quarantines, biologic aspects, 435
 - disinfestation, 437
 - economic aspects, 436
 - efficacy of, 440
 - embargoes, 437
 - Federal, affecting trade, 438
 - inspection, 437
 - mechanism of, 437
 - permits, special, 438
 - present-day, 438
 - shipment, unrestricted, 437
 - state-imposed, affecting interstate trade, 439

R

- Raleigh, Sir Walter, and introduction of the potato to Europe, 3
- Raspberry, cane fruit mosaic, 300
- Red rot of sugar cane, 112
 - control, 113
- Red rust, of wheat, 27
- Reduction in population of available host plants, 427
- Resistance in control of plant diseases, 442-456
 - acquired immunity, 448
 - basic concepts of plant immunology, 443-445
 - chemical defenses, 446
 - complexity of factors, 450
 - economic significance, 442
 - functional defenses, 447
 - hypersensitivity, 448
 - mechanical defenses, 445
 - nature of, 445-450
- Resistant varieties in control of plant diseases, 450-456. *See also individual diseases*
- Rhizoctonia*, 74

Rhizoctonia—(Continued)

- disease of potatoes, 88-93, 90
 - control, 91
 - cultural practices, 93
 - seed-tuber treatments, 92
 - use of healthy seed tubers, 91
 - epiphytology, 91
 - etiology, 89
 - history and distribution, 88
 - host plants, 89
 - importance, 88
 - symptoms and signs, 89, 90
- solani*, 88-93, 90, 238-244, 244, 247, 248, 251, 259
- Rhizomorphs, 16
- Rhizopus*, 254
 - nigricans*, 211, 225-229, 227, 245, 247, 248
 - tritici*, 227
- Rhubarb, *Verticillium* wilt, 155
- Ribes*, blister rust, 43
- Rice, brown spot, 180
 - Cercospora* leaf spot, 186
- Rimocortius psorosis*, 327
- Robigus, rust god of the Romans, 2
- Roguing, 480
- Root knot, 353-361, 355, 356
 - control, 357
 - in commercial vegetable production, 359
 - in field crops, 357
 - in fruit orchards, 357
 - in greenhouse and seedbeds, 361
 - in home vegetable gardens, 359
 - in nurseries, 358
 - in ornamental plantings, 360
 - distribution and importance, 353
 - epiphytology, 355
 - etiology, 354
 - host plants, 353
 - symptoms and signs, 354, 355
- Root lesion nematode, 364, 365
- Root rot, 156-177
 - black, of tobacco, 417
 - Rhizoctonia*, 417
 - Texas. *See* Texas root rot
- Rose(s), black spot, 137, 138
 - cankers, 201-203, 202
 - powdery mildew, 126, 127
- Rosette, of apple, 373
 - of pecan, 372, 372
- Rot. *See* Bacterial ring rot, Black rot, Bitter rot, Blossom-end rot, Boll rot, Brown rot, Charcoal rot, Ear rot, Neck rot, Red rot, Root rot, Shoestring root rot, Soil rot, Sorghum root and stalk rots, Stalk rot, Texas root rot, White trunk rot, etc.

- Rotation, for bacterial blight of cotton, 277
 for bacterial plant diseases, 264
 for cereal anthracnose, 191
 for clubroot of crucifers, 235
 in control of plant diseases, 458
 for downy mildews of grains and grasses, 222
 in dry-land foot or root rot of wheat, 176
 for Texas root rot, 162
- Rotenone, for smut of corn, 71
- Ruga verrucosans*, 323-325, 323
- Rugose mosaic of potato, 329, 330
- Rust(s). *See* Bean rust, Leaf rust, Red rust, Stem rust, White rust, etc.
 alfalfa, 40
 black of wheat, 27
 blister, of five-needle pines, 43, 44
 carnation, 47, 47
 as cause of plant diseases, 22-48
 of cereals, 8
 of corn, 39
 cotton, 371
 of field crops, 23-40
 fungi, 22
 races, physiologic, 22
 specialization, physiologic, 22
 of grasses, 40, 41
 heteroecious, 22
 hollyhock, 47, 48
 monoecious, 22
 orange, of cane fruits, 45
 of ornamentals, 47-48
 stem, black. *See* Stem rust
 of stone fruits, 45
 of trees and shrubs, 41-45
 of vegetables, 45-46
 of wheat, leaf. *See* Leaf rust of wheat
 white, of crucifers, 223, 225
- Rust-resistant varieties as a control measure. *See* individual diseases
- Rye, leaf rust, 39
 nematode, 362, 363
 smut, 60

S

- Sanitation, for bacterial plant diseases, 264
 for cereal anthracnose, 191
 for clubroot of crucifers, 235
 in control of plant diseases, 458
 for crown gall, 295
 for tobacco mosaic, 315
- Saprogenesis, 424
- Saprolegniales*, 211
- Saprophytes, soil, 264
- Scab, apple, 99-105, 100, 431. *See also* Apple scab

Scab—(Continued)

- of cereals, 95-99
 control, 98
 disease-free seed, 98
 rotation, 98
 resistant varieties, 99
 sanitation, 98
 epiphytology, 98
 etiology, 97
 history and distribution, 95
 host plants, 96
 importance, 95
 seedling-blight stage, 413, 413
 symptoms and signs, 96, 96, 97
 common, of potato, 294-296, 295, 417
 control, 296
 epiphytology, 296
 etiology, 295
 symptoms, 295
 of peach, 184, 184
- Scald, of apple, 382, 383
- Scalding of bean, cowpea, and soybean leaves, 392
- Scaly bark, 327-329, 328. *See also* Citrus psoriasis
- Sclerospora*, 222
farlowii, 222
- Sclerotium(a), 16
- Seed, as carriers of viruses, 306
 certified, 467
 disinfested, 468-476
 indexed, 467
 noninfested, 466-476
 plant parasites, 341-351
 state-certified, in control of plant diseases, 440
 treatment, 469
 for bacterial blight of cotton, 277, 278
 for bacterial diseases of cereals, 280
 for bacterial wilt of corn, 284
 for cereal anthracnose, 191
 for corn root, stalk, and ear rots, 256
 for cotton seedling blight and boll rot complex, 249, 251
 for damping-off, 241
 dry heat, 475
 hot water, 475
 vapor heat, 476
 uninfested, 467
 certified, 467
 cleaned, 467
 indexed, 467
 from protected seed blocks, 467
 selected, 467
 from uninfested areas, 467
- Seed dusting, for cotton seedling blight and boll rot complex, 252
 limitations, 251

- Seeding, in control of plant diseases, 477
- Seedling infection smuts, 50-60
- Semesan, 472
for clubroot of crucifers, 236
for damping-off, 242
- Semesan Bel, 472
for black scurf of potato, 92
for crown gall, 294
- Semesan, Jr., 471
for corn root, stalk, and ear rots, 257
- Septoria apii-graveolentis*, 208, 208
diseases of wheat, 203-206
leaf spot of tomato, 206-208, 207
control, 207
lycopersici, 206-208, 207
tritici, 203, 204
- Shoestring root rot, 81-86
control, 85
epiphytology, 84
etiology, 84
history and distribution, 81
host plants, 81
importance, 81
symptoms and signs, 82, 83
- Shothole, cherry, 134, 135
- Shrubs, rusts, 41-45
- Signs of plant diseases. *See individual diseases*
- Slime molds, 236, 237
- Smudge, onion, 195
- Smut(s), barley, 57
blossom infection, 50, 61-67, 431
as cause of plant diseases, 49-72
common, of corn, 67-71
control, 71
rotation, 71
sanitation, 71
epiphytology, 69
etiology, 69
history and distribution, 67
host plants, 67
importance, 67
symptoms and signs, 68, 68
control, 40
corn, chlamydospores, germinating, 70
of barley, 66
of sorghum, 58
of wheat. *See Bunt of wheat*
flag, of wheat, 59
local infection, 50, 67-72
loose, brown, of barley, 66
of sorghum, 58
of wheat, 61-66
control, 63
hot water seed treatment, 63, 64
machine for, 65, 66
epiphytology, 62
- Smut(s)—(Continued)
loose—(Continued)
of wheat—(Continued)
etiology, 62
history and distribution, 61
host plants, 61
importance, 61
symptoms and signs, 62
nature of, 49-50
oats, formaldehyde treatment, 57, 59
onion, 72, 72
rye, 60
seedling infection, 50-60
barley, 57
bunt of wheat, 50-57
oats, 57, 58
sorghum, 58, 60
stinking. *See Bunt of wheat*
types of, characteristics, 50
white, 72
- Snapdragon, root knot, 355
rust, 47, 47
- Soft rot, sweet potato, and strawberry leak, 225-229, 227
control, 228
sanitation, 228
storage conditions, 228
varietal resistance, 229
epiphytology, 228
etiology, 227
history and distribution, 225
host plants, 226
importance, 226
symptoms and signs, 226
- Soft rot of vegetables, 272
control, 273
- Soil, acid, contagious diseases favored by, 416
alkaline, contagious diseases favored by, 417
amendment, in control of plant diseases, 476
disinfestation, for clubroot of crucifers, 235
for damping-off, 239, 242
disinfested, 459-466
by heat, 459-463, 460, 461
by steam, 459
excess of chemicals, as cause of plant disease, 375-377
fumigation, in control of plant diseases, 463-466
nemas, 464
noninfested, 457-466
in parasitic disease, 415
fertility, 415
N-P-K ratio, 415
reaction, 416, 416

Soil—(Continued)

- poisoning by sprays, 487
- preparation of, in control of plant diseases, 476
- rot, of sweet potato, 417
- texture, effect on plant diseases, 417
- in transmission of viruses, 307
- unfavorable conditions as cause of plant diseases, 368-381
- uninfested, 457-459
 - fallowing, 458
 - new land, 457
 - rotation, 458
 - sanitation, 458
 - barriers, 458
- waterlogged, as cause of plant disease, 381
- Sorghum(s), blasting, 381
 - leaf rust, 39
 - leaf spot, 279, 280
 - leaf streak, 279, 280
 - leaf stripe, 279
 - root and stalk rots, 257-261, 258
 - control, 260
 - smut fungi, 58, 60
- Southern wilt, 289
 - control, 290
 - etiology, 290
 - symptoms, 289
- Soybeans, downy mildew, 223
 - scalding of leaves, 392
- Spartina pectinata*, 41
- Specimens, laboratory examination of, for study of plant diseases, 397
 - preservation of, for study of plant diseases, 397
- Speck, bacterial, of tomatoes, 273, 274
- Speckled leaf blotch of wheat, 203, 204
 - control, 205
- Spergon, 469, 473
 - for black rot, of sweet potato, 115
 - for black scurf of potato, 92
 - for bunt of wheat, 54
 - for corn root, stalk, and ear rots, 257
 - for cotton seedling blight and boll rot complex, 250
 - for damping-off, 242
 - for smut, of sorghum, 59
- Spermagonia, 26
- Sphacelotheca cruenta*, 58
 - sorgbi*, 58
- Sphaeriales*, 94
- Sphaeriopsidales* as cause of plant diseases, 196-208
- Sphaerotheca humuli*, 126
 - pannosa*, 126, 127
- Sporangiospores, 211
- Spores, 16

Spores—(Continued)

- germination of, methods of studying, 398
- Spindle tuber virus, of potato, 331, 331
- Sporidia, 49
- Sporodochia, 94
- Spot, bacterial, of tomatoes, 273, 274
 - blotch, barley, from *Helminthosporium sativum*, 178, 179
 - brown, of corn, 229-232, 230, 231
 - of rice, 180
 - Kelsey, of plums, 386, 387
 - leaf, of alfalfa, 136
 - angular, of tobacco, 275
 - of beets, 186, 187
 - of celery, 187
 - Cercospora*, of peanut, 185, 186
 - of cherry, 134, 135
 - of elm, 110, 111
 - of iris, 113, 113
 - sorghum, 279, 280
 - white, of alfalfa, 381
- Spraying, 482, 483-485, 484
 - for anthracnose, of beans, 195
 - of cane fruits, 137
 - melon, 194
 - sycamore, 112
 - for bacterial plant diseases, 264
 - for bacterial spot of stone fruits, 270
 - for bitter rot, apple, 108
 - for black knot of plums and cherries, 110
 - for black rot, apple, 106
 - grape, 109
 - for black spot, of roses, 138
 - for blight, early, of potato and tomato, 182
 - late, of potato and tomato, 216, 484
 - for blotch, of apple, 200
 - for brown rot of stone fruits, 134
 - for canker, crown, of roses, 203
 - for cherry leaf spot, 135
 - for downy mildew, 222
 - of tobacco, 221
 - vs. dusting, 481
 - for elm leaf spot, 111
 - for fire blight of apple and pear, 268
 - for grape black rot, 109
 - hazards, 486-488
 - injury, 486
 - for iris leaf spot, 114
 - for leaf curl, peach, 141
 - for leaf spot, cherry, 135
 - elm, 111
 - iris, 114
 - for peach leaf curl, 141
 - for powdery mildew, of apple, 129
 - of cucurbits, 129
 - of roses, 127
 - for rust, cedar apple, 43

- Spraying—(Continued)
 for scab, of apple, 102–105
 peach, 185
 for *Septoria* leaf spot of tomato, 208
 for tomato leaf mold, 184
 for white rust of crucifers, 224
 Sprays, 482, 483–485, 484
 copper series, 483
 incompatibility, 487
 methods of application, 485
 organic series, 485
 poisoning of soil by, 487
 as cause of plant disease, 377
 residues, 487
 sulfur series, 483
 Spinach, blue mold, 8
 downy mildew, 218, 219
 white rust, 8, 224
 Steam, in disinfection of soil, 459
 for root knot, 360
 Stem, canker, 201
 cracked, of celery, 373
 nematode, 363
 Stem rust, on barberry common, 24, 29, 30
 of barley, 29
 of grains and grasses, 23–40
 control, 31
 agronomic practices, 33
 eradication of barberry, 33
 resistant varieties of wheat, 31
 sulfur dusting, 33, 34
 epiphytology, 31
 etiology, 27
 history and distribution, 23
 host plants, 24
 importance of, 23
 life cycle, 27, 27
 symptoms and signs, 24
 of oats, life cycle, 28
 of rye, 29
 spores, types of, 30
 of wheat, 25
 losses from, 24
 sources of infection, 28
 work of de Bary, 23
 Sterilization of soil, for black root rot of tobacco, 169
 Stewart's disease, of corn, 282–285, 282–284
 Stone fruits, bacterial spot, 268–270, 268
 rust, 45
 Verticillium wilt, 155
 virus diseases, 334–338
 Storing, in control of plant diseases, 478
 Strawberry, leak, 225–229
 Verticillium wilt, 155
 Streak, leaf, of sorghum, 279, 280
 Sireptomycetes scabies, 294
 Striga lutea, 350
 Stripe rust of wheat, 38
 control, 38
 history and distribution, 38
 plant hosts, 38
 symptoms and signs, 38
 Sugar beet, curly top, 323–325, 323, 431.
 See also Curly top of sugar beet
 dry rot, 373
 nematode, 361, 362, 422
 Sugar cane, mosaic, 319
 red rot, 112
 Sulfur, wettable, for scab, common, of potato, 296
 for spraying, 483
 Sulfur dioxide injury to plants, 383, 384
 Sulfur dusting, for leaf rust of wheat, 37
 for powdery mildew, of cereals, 126
 of cucurbits, 128
 of legumes, 128
 of roses, 127
 for stem rust of grains and grasses, 33, 34
 Sulfuric acid, in control of plant diseases, 466
 Sunflower rust, 40
 Sunstroke of plants, 386
 Surveys, by airplane, 396
 of plant disease, 394–397, 396
 Sweet corn, bacterial wilt. *See* Corn
 Sweet potato, black rot, 114, 115
 control, 114
 pox, 417
 rust, 46
 soft rot, 225–229, 227
 soil rot, 417
 wilt or stem rot, 151, 152
 control, 151
 Sycamore anthracnose, 112, 112
 control, 112
 Symptoms of plant diseases. *See individual diseases.*
- T**
- Taphrina deformans*, 138–141, 139
Taphrinales, 94
 Tear gas, for black root rot of tobacco, 169
 for damping-off, 239, 242
 for root knot, 360
 Teliospores, 26, 26
 Temperature, effect on parasitic disease, 413, 413
 unfavorable, as cause of plant diseases, 386–390
 Tersan, 466, 474
 Tersan, for black rot, of sweet potato, 115
 for black scurf of potato, 92

Tersan—(Continued)

- for blight, early, of tomato, 182
- for brown rot of stone fruits, 134
- for damping-off, 242
- for scab, common, of potato, 296
- for smut of onion, 72

Tetrachlorethane, for root knot, 360

Tetramethylthiuram disulfide, for brown rot of stone fruits, 134

Texas root rot, 8, 156–166, 417

- control, 162
 - avoidance of susceptible perennials, 163
 - barriers, 163, 163
 - eradication, 163
 - manuring, 164, 165
 - ornamental and fruit trees, 165
 - rotation, 162
 - vegetables, 166
 - weeds, 165
- epiphytology, 161
- etiology, 161, 162
- history and distribution, 156
- host plants, 158
- importance, 156
- symptoms and signs, 8, 159, 157–160

Theophrastus, 2

Therapeutic treatments of trees, 490

Thielavia basicola, 167*Thielaviopsis basicola*, 166–169, 168*Tillandsia usneoides*, 351*Tilletia*, 49

- caries*, 50–57
- foetida*, 50–57

Tipburn of potatoes, 387, 388

Tobacco, angular leaf spot and wildfire, 275

- downy mildew, 220, 220, 221

Tobacco, mosaic, 305, 305, 313–316, 314

- control, 315
 - breeding for resistance, 316
 - sanitation, 315
- etiology, 315
- history and distribution, 313
- host plants, 314
- importance, 314
- symptoms, 314

- root rot, black, 166–169, 168, 417

Tomato, anthracnose, 195

- bacterial canker, spot, and speck, 273, 274

- blight, early, 181–184

- blossom-end rot, 379, 380

- leaf mold, 183, 184

- control, 184

- phosphorus deficiency, 370

- Septoria* leaf spot, 206–208, 207

- Verticillium* wilt, 155

Tomato—(Continued)

- wilt, 148, 416

- control, 149

- symptoms, 149

Toxic trace elements, as cause of plant disease, 377

Tranzschelia pruni-spinosae, 45

Trees, fruit, control of Texas root rot in, 165

- ornamental, control of Texas root rot in, 165

- rusts, 41–45

- therapeutic treatments, 490

Tulip, virus disease of, 299

Turnips, brown heart, 373

U

Uredinales, 22

Urediospores, 26, 26

Urocystis, 49

- agropyri*, 59

- cepulae*, 72, 72

- occulta*, 60

Uromyces caryophyllinus, 47, 47

- elegans*, 40

- phaseoli*, 46, 46

- striatus*, 40

Ustilago, 49

- avenae*, 58

- hordei*, 66

- levis*, 58

- nigra*, 66

- nuda*, 66

- tritici*, 61–66

- zeae*, 67–71

V

Vasco-4, for damping-off, 242

Vegetables, control of Texas root rot in, 166

- rusts, 45–46

- soft rot, 272

Vein-banding virus, of potato, 329

Venturia inaequalis, 99–105, 100

Vertebrates, as cause of plant disease, 15

Verticillium, 142, 143

- albo-atrum*, 155

- wilt, 148, 155

- beet, 155

- bush fruits, 155

- of cotton, 155

- of cucumber, 155

- of eggplant, 490

- of hops, 155

- of maple, 155, 490

- of muskmelon, 155

- Verticillium*—(Continued)
 wilt—(Continued)
 of okra, 155
 of peppers, 155
 of potato, 155
 of rhubarb, 155
 of tomato, 155
 Victoria blight, of oats, 180
 Violet, downy mildew, 222
 Virosis, yellow-red, 337
 Virus(es), as cause of plant diseases, 15,
 298–338
 heat treatment, 490
 leaf roll, of potato, 331, 332
 mosaic, latent, of potato, 329
 mild, of potato, 330
 nature of, 311–313
 nomenclature, 311
 potato, "healthy," 329
 properties, 303–311
 host ranges, 307
 immunity, acquired, 309, 309, 310
 infectiousness, 303
 mutability, 308
 number, 305
 obligate parasitism, 303
 resistance, to chemicals, 308
 to heat, 308
 retention of infectivity in storage, 308
 serologic reactions, 309
 size and shape, 304, 304
 transmission, 306
 by dodder, 307
 by insects, 306
 by mechanical means, 306
 by seed, 307
 by soil, 307
 by vegetative propagation, 307
 spindle tuber, of potato, 331, 331
 of stone fruits, control, 337
 vein-banding, of potato, 329
 Virus disease(s), in cowpea, 301
 inoculation methods, 403
 in Jimson weed, 301
 of potatoes, 329–334
 control, 333
 field practices, 334
 resistant varieties, 334
 seed inspection, 334
 use of virus-free seed tubers, 333
 significance of, 298–300
 specific, 313–338
 of stone fruits, 334–338
 symptoms, 300–303
 combinations, 303
 distortions, 302
 growth, abnormal, 302, 302
 reduced, 302
 Virus disease(s)—(Continued)
 symptoms—(Continued)
 hypertrophy, 302
 inhibition of pigment formation, 301
 anthocyanin, 302
 chlorophyll, 301
 internal, 303
 necrosis, 303
 of tulips, 299
- W**
- Wallrothiella arceuthobii*, 350
 Water, deficiency of, as cause of plant dis-
 ease, 377–379
 excess of, as cause of plant disease,
 379–381
 supply, in control of plant diseases, 477
 irregular as cause of plant disease,
 379–381
 Watermelon, *Verticillium* wilt, 155
 wilt, 7, 7, 149
 control, 150
 work of Florida Experiment Station,
 11
 Weather, relation to plant diseases, 404
 Weed(s), control by clean culture, 478
 for control of Texas root rot, 165
 Wheat, anthracnose, 188–192, 190
 black chaff, 281, 282
 black rust, 27
 bunt of, 50–57, 52, 414
 diseases of, 9
 dry-land foot or root rot, 169–177, 172
 glume blotch, 205, 206
 leaf rust, 34–37, 36, 431
 loose smut, 61–66, 52
 mosaic, 318, 318
 nematode, 362, 363
 red rust, 27
 root rot, 169–177, 172
 scab, 96, 97
 Septoria diseases, 203–206, 204
 speckled leaf blotch, 203, 204
 stripe rust, 38
 yellow berry, 368, 369
 White rust, 223–225
 of crucifers, 223, 225
 control, 224
 of mustard, 224
 of spinach, 8, 224
 White smuts, 72
 White spot, of alfalfa, 381
 White trunk rot, 76–81, 78, 80
 control, 79
 with decay-retarding wood preserva-
 tives, 81
 in forests, 79

White trunk rot—(Continued)**control—(Continued)**

in individual ornamental or fruit trees,
79

in woodlots or other small managed
plantings, 79

distribution, 76

epiphytology, 79

etiology, 77

host plants, 76

importance, 76

symptoms and signs, 77

Wildfire, of tobacco, 275

Wilt, alfalfa, 285–287, **286**. *See also* Alfalfa,
wilt

bacterial, 282–291. *See also* Bacterial wilt
Cephalosporium. *See Cephalosporium* wilt

cotton, 143–148, **145**. *See also* Cotton wilt
cowpea, 154

Wilt, diseases, 142, **143**

Fusarium, in cabbage, 443

pea, 154

purpletop, of potato, 7

tomato, 148, 416

Verticillium. *See Verticillium* wilt

Wind, in parasitic disease, 415

Winter browning of evergreens, 390

Winter injuries to plants, 390

Winterkilling, of plants, 388

Witchweed, 350

control, 351

Wood, decay, 74–86

losses from, 76

registered propagation, 468

X

Xanthomonas holcicola, 279

malvacearum, 275–279, **276**

phaseoli, 270–272, **271**, **272**

pruni, 268–270, **268**

Xanthomonas holcicola—(Continued)

translucens, 279, 282

vesicatoria, 275

vignicola, 272, **273**

X-disease of peach, 337, 490

X-virus of European workers, 329

Xylene for root knot, 360

Y

Yellow berry, of wheat, 368, **369**

Yellow dwarf, of potato, 7

Yellow mosaic, **318**

Yellows, alfalfa, 373, **373**

aster, 321–323, **322**. *See also* Aster yellows
peach, **302**, 335

Y-virus of European workers, 329

Z

Zerlate, 474, 485, 487

for anthracnose, tomato, 196

for bitter rot, apple, 108

for black rot, sweet potato, 115

for blight, early, of potato, 182

for brown rot of stone fruits, 129

for leaf spot, cherry, 135

Zimate, 474

for bitter rot, apple, 108

for blight, early, of potato, 182

Zinc, deficiency, as cause of plant diseases,
372, **372**, **375**

Zinc dimethyl dithiocarbamate, 108, 474

for anthracnose, tomato, 196

for blight, early, of potato and tomato,
182

Zincate, 474

Zoösporangia, **211**

Zoöspores, **211**

Zygospore, 210, **211**

Zygote, 210

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